PRINCIPLES AND PRACTICE

OF

INFANT FEEDING

JULIUS H. HESS, M.D.

Principles and Practice of Infant Feeding

BY

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ILLUSTRATED

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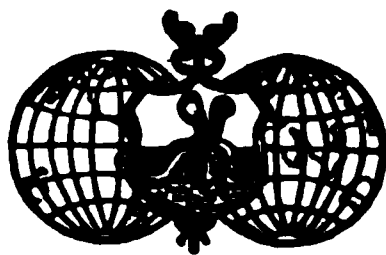
TO

ISAAC A. ABT, M.D.

MY FRIEND AND TEACHER

THIS BOOK IS AFFECTIONATELY DEDICATED

PREFACE TO THIRD EDITION.

In the preparation of the third edition the chapters on Vomiting, Colic and Flatulence, Constipation, and Abnormal Stools have been completely rewritten.

The classification, nomenclature and pathogenesis of the nutritional disturbances have been revised to conform to the latest researches. American clinics are to be credited with the greater part of this progress.

New chapters dealing with rickets, scurvy, spasmophilia, acidosis and anemias of infancy have been added.

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PREFACE.

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It has been our experience that the best results obtained in the teaching of the principles and practice of infant feeding have been accomplished when the theory of feeding and the study of actual cases have been combined.

Our object in publishing this volume is to place in the hands of teachers and students a manual on infant feeding to be used in preparation for clinical conferences. Whenever possible, the subject under discussion is illustrated in the class-room by clinical cases and case records from the teacher's personal material.

While there are many excellent works covering this subject, we have found most of them to be too voluminous to fulfill our needs, and we have therefore attempted to present the subject in concise form in this small volume.

For the teaching of nurses we have selected those chapters which have to do with the nursing care of premature, healthy, and sick infants, the feeding of breast-fed and artifically fed healthy babies, and the preparation of infants' foods and diets.

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INTRODUCTION.

THE dependence of the offspring upon its mother for food to supply its primitive needs can only be realized when we remember that one-fourth of the civilized race die during the first year of life, and that 60 per cent. of these deaths are due to nutritional disturbances, while a large portion of the other 40 per cent. are primarily dependent upon impairment of the infant's constitution by improper feeding. The mortality of the first year is nearly 60 times that of the fifteenth year, and it is not until we approach the 85th year that we meet with such a high percentage death-rate. The problem is not simply to save life during the perilous first year but to adopt those means which shall tend to healthy growth and normal development. The child must be fed not only to avoid the immediate dangers of acute indigestion, diarrhea, and marasmus, but the more remote ones—rickets, scurvy, and general malnutrition. These latter three are the most important conditions that predispose to disease in early life.

A growing child requires far more food than its weight would indicate. For, in the first place, its intake must exceed its expenditure, so that it may grow. The expenditure of an organism is pretty nearly in proportion, not to its mass, but to its surface. The skin surface of a boy from 6 to 9 years, with a body weight of 18 to 24 kilograms (40 to 50 pounds), is two-fifths to one-half that of a man of 70 kilograms (154 pounds), and he

should therefore have about half as much food as the man. This disproportion in the needs of the infant as compared with the adult, is even greater than that of the child compared with the adult. By exact measurements it has been determined that an infant from its fourth to the sixth month consumes about twice as much food per kilogram body weight as the adult.

PART I.

General Considerations.

CHAPTER I.

THE ANATOMY OF THE DIGESTIVE TRACT OF THE INFANT.

Oral Cavity. The salivary glands are well developed at birth, and the active principles of the salivary secretion are present, but in small quantities. Teething begins at about the sixth month, and dentition is not completed until about the end of the second year. In most instances this is a normal physiological process, and should cause no disturbances. However, in a considerable number of cases the gastric and intestinal secretions are affected reflexly, with a diminished activity on the part of these glands; and if there is any tendency to a general disturbance during this period, a reduction in the quantity of the food administered is indicated. However, far too great an importance is usually given by the laity to the process of teething.

Stomach. In the newborn the stomach has a more vertical position than in the adult. However, röntgenologic examination has demonstrated that it is less vertical than has been formerly supposed. The cardiac end is found at the left of the tenth dorsal vertebra. The pylorus lies about midway between the ensiform cartilage and the umbilicus. The position of the stomach and its form, due to lack of development of the fundus and lack of muscular development at the cardiac end, account in great part for the frequency of vomiting in the infant.

The **pylorus** also lacks the muscular development of the adult, and is decidedly more patent.

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Considerable difficulty is experienced in our attempts to gain accurate knowledge of the capacity of the stomach. Pfaundler, who measured the size of the stomach in numerous infants, using air under a given pressure, has given us figures which are, in all probability, fairly accurate.

He states that the capacity at birth is 2 ounces (60 mils), at one month 2 to 3 ounces (60 to 90 mils), at six months 6 ounces (180 mils), and at one year 9 to 10 ounces (270 to 300 mils). The importance of the stomach's capacity in determining the size of the individual feeding is only relative, dependent to a great extent upon the form of diet. With milk as the food, a considerable portion of the water content passes through the pylorus before the meal is finished, if the food is not too rapidly given. When a child is fed by gavage, the size of the meal is of greater importance because of the danger of overdistention by the rapid administration of the food by this method. Notwithstanding the fact that the size of the stomach varies in different babies, we have found it a good working rule with normal infants to administer at each feeding a quantity 2 ounces more of the liquid food than the infant is months old.

The intestines are relatively larger than in the adult, which applies more especially to the large intestine, and particularly to the sigmoid flexure. The sigmoid is also more mobile, due to the greater length of the mesosigmoid, and is extra-pelvic. The musculature is relatively thin, and bears an important relationship to the frequency of intestinal distention and the presence of colic, which is due to the stagnation of large quantities of gas in the intestinal tract.

The pancreas shows no special anatomical differences.

The liver is relatively two-and-a-half times as large at birth as in the adult, and is easily palpable, and in the nipple-line of the right side usually extends 1 to 1½ inches (2 to 4 cm.) below the costal border.

CHAPTER II.

THE PHYSIOLOGY OF THE DIGESTIVE TRACT OF THE INFANT.

WHILE all the ferments are present in early life, they vary quantitatively and qualitatively as compared with older children.

Mouth. Ptyalin, which is an amylolytic ferment, is present in the saliva immediately after birth, but is small in amount, and weak in its action. Albumin, water and mucus in saliva vary with the variety of food taken (Pavlow).

Stomach. Gastric juice is present in the stomach even in the premature. Its secretion is mainly stimulated by the act of sucking and by the presence of the food in the stomach.

Free hydrochloric acid is little less than in the adult. It may be stated that the small protein content of human milk, as compared with cow's milk, favors the presence of hydrochloric acid. This is a point of great importance in the food problem of the infant. Free hydrochloric acid is found in 10 per cent. of cases after 1 hour, and in 33 per cent. of cases after 1½ hours on feeding with human milk (Hamburger and Sperck). With cow's milk, free hydrochloric acid is found very rarely, which is due to combination of the hydrochloric acid with salts and proteins. Total acidity is in small part only due to free hydrochloric acid. More important are phosphoric acid, acid phosphates, acid chlorides, fatty acids and acid albumins (albumoses and peptones). Total acidity is 20 to 60 mils N :10 acid to 100 mils of gastric contents. The action of the hydrochloric acid is as follows: (1) it makes protein digestion possible (acid albumins); (2)

stimulates the pancreas; (3) disinfects and exerts anti-toxic action.

The following ferments are present in the stomach: (1) *Pepsin*, which is present at birth, and is active and causes at least partial digestion of proteins. It increases to the fourth month, then remains fairly constant. More pepsin is present in bottle-fed infants. (2) *Rennin* is also present at birth, and in the presence of hydrochloric acid coagulates milk. Whether this is dependent on pepsin, or whether it is a specific ferment, is a question. (3) *Lipase*, a fat-splitting ferment, is found in the stomach in small quantities, and is probably a definite product of the gastric mucosa.

Small Intestines. Mucous membrane of the small intestines secretes about 1 liter of juice daily, and this contains all ferments at birth, they being, however, relatively feeble at first. The following ferments are present in the intestinal secretion: (1) *erepsin* (Cohnheim), which splits casein, albumoses, and peptones to peptids and amino-acids. Other albuminous bodies are not affected by it. (2) *lactase, maltase, invertin;* they split disaccharides (milk, malt, and cane sugar) to monosaccharides, and each is stimulated by its own sugar. (3) *prosecretin*, which is changed to secretin by hydrochloric acid from the stomach, and stimulates the secretion of the pancreas. (4) *enterokinase*, which activates the proteolytic enzyme of the pancreatic juice; and probably (5) *diastase*.

Pancreas. All of the ferments (trypsin, steapsin, and amylopsin) are found in the intestines at birth.

The liver possesses the ability to form glycogen and urea in the newborn. Bile is present, its emptying from the gall-bladder being stimulated by chemical action of fats on the duodenal mucous membrane. The functions of the bile are: (1) to hold fatty acids and fatty acid salts in solution, (2) to stimulate the pancreas, and (3)

an antiseptic action. Other functions of the liver are formation of urea, and formation and storing of glycogen.

Large intestines secrete no enzymes, their chief function being absorption of water and throwing off of Ca, P, Na, K, Fe, Mg.

CHAPTER III.

METABOLISM IN INFANTS.

1. General Considerations.

THE term metabolism covers all of the functions of the human body which have to do with the preparation for and assimilation of food.

To furnish the body with fuel for its normal activities, the following groups of food elements are necessary: proteins, fats, carbohydrates, salts, and water. Fats and carbohydrates, and to a lesser extent proteins, furnish fuel; while the proteins and salts more especially form the elements necessary for body growth.

It is necessary to distinguish between the activities which take place within the gastro-intestinal tract before absorption of the changed products and the deeper seated metabolism which takes place beyond the intestinal wall, which can be designated as the "intermediary metabolism."

Under normal conditions in the adults the intake and the products of excretion balance one another, while in the infant there is a positive balance—that is, less is excreted than is absorbed—and one may well say that a balance which would be normal in the adult is pathological in the child, and would thereupon soon result in a stationary weight, or a loss in weight.

Several factors offer difficulties in the study of infant metabolism.

First, it is difficult to obtain stools free from urine and with the water content intact.

Secondly, the small volume in which the urine and stools are obtained offers many difficulties in their study.

Urine and stool examinations should cover a period of at least three days to be of conclusive value.

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2. Composition of Milk and the Metabolism of Its Constituents.

The natural food of the infant is *human milk*, characterized by the fact that its quality changes very little, the infant's growth being dependent on the increase in amount of milk secreted.

Milk of different animals varies as to its fuel value, and also in its chemical composition, especially quantitatively there being marked differences.

	Protein	Fat	Sugar	Salts
Human	1.5	3.5	6 to 7	0.20 per cent.
Cow's	3.4	3.8	4 to 5	0.75 " "

Human *colostrum* differs from the mature human milk in that the protein and salts are higher. Protein averaging 2.25 per cent., and the average ash 0.3077. Sugar not differing greatly from that the later milk, but averaging somewhat lower. Fats are also somewhat lower, averaging 3.15 per cent. (Holt[1]). These figures vary in different women, and also with the day of puerperium, but represent average specimens. Colostrum also contains numerous leucocytes, and large cells containing fat, these latter probably being epithelial in origin.

1. **Proteins.** *Chemistry of Proteins.* Proteins contain carbon, hydrogen, nitrogen, oxygen, sulphur, and phosphorus. They are highly complex chemical substances, similar in their chemical composition to protoplasm and essential to life.

Of the proteins milk contains mainly casein and albumins, with small amounts of globulins, opalisin, nuclein, etc.

100 mils of milk contain	Albumin	Casein
Human milk	0.6 Gm.	0.8 Gm.
Cow's milk	0.2 to 0.3 Gm.	2.7 to 3.0 Gm.

Casein belongs to the nucleo-albumin group (proteins), which contain phosphorus, are insoluble in water, mod-

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[1] Amer. J. Dis. Child., Vol. X, p. 228, October, 1915.

erately in alkalies, precipitated by acids, not coagulated by boiling, and by pepsin digestion changed to para- or pseudo- nucleins (which are bodies rich in phosphorus). Chemically it is composed of a complex group of amino-acids, the basis of all protein bodies, and a prosthetic group which contains the phosphorus. Amino-acids are characterized by the group COOH, in which an H is replaced by NH_2 group, *e.g.*, acetic acid (CH_2HCOOH), amino-acetic acid, or glykokoll (CH_2NH_2COOH).

Human casein contains much less phosphorus than cow's (0.25 to 0.88). This proves that the casein of the human and the casein of the cow's milk are different bodies, although this difference is probably of a quantitative nature only. The two caseins differ also in their coagulability, the human casein being more difficult to precipitate with acids, salts and rennin. The soluble albumins are coagulated by heat and weak acids.

Metabolism of Proteins. Casein is separated from the so-called whey albumin, and is changed to an insoluble paranuclein. It is unknown whether or not the enzyme causing it is identical with the protein digestive ferment secreted by the gastric mucous membrane.

Pepsin (from the pyloric mucous membrane) changes paranucleins to albumoses and peptones, which then pass into the small intestines. (Erepsin, the ferment of the intestinal juices, works very rapidly on the end products of pepsin digestion.) In the small intestine an intricate splitting takes place.

With the human milk as a food, a very small amount of nitrogenous products of the food appears in the stools, the total being about one-sixth of the intake, and part of this arises from

      1. Intestinal juices,
      2. Intestinal epithelium.
      3. Bacterial activity.

After passing through the intestinal wall, proteins have three functions to perform;

1. To replace used proteins (lost through urine, sweat, digestive juices, cell destruction, etc.).

2. To satisfy cell growth which would be impossible without proteins.

3. To furnish fuel for part of the dynamic loss (fats and carbohydrates are the natural fuels, the protein combustion being of secondary importance).

In the average feeding with cow's milk, three times as much protein is given as needed for 1 and 2, therefore it is used for 3, (that is, dynamic purpose), or is rejected by the body.

The great disproportion as seen in a comparison of the proteins in cow's milk over human milk is probably due to the needs for cell growth in the calf. Due to the ability of, the organism, within certain limits to regulate its function, the excess of protein in the average diet with cow's milk as a basis is excreted by way of the intestinal tract. Cell growth is therefore not excessively stimulated on these relatively high protein diets.

End Products of Protein Metabolism in Urine:

Urea 60 to 80 per cent. Ammonia 3 to 10 per cent.

Oxaluric bodies ⎫
Uric acid · ⎬ Nitrogenous by-products.
Kreatinin ⎪
Oxybutyric acid ⎭

Urea forms 75 to 86 per cent. of the nitrogen constituents of the urine.

By *ammonia coefficient* is meant the relation of ammonia to the other nitrogenous bodies in the urine.

Influence of the Carbohydrates and Fats on the Nitrogen Metabolism.

1. Carbohydrates cause
    (1) Increased retention of proteins.
    (2) Increased nitrogen in feces.

2. Fats cause
    (1) No increased protein retention.
    (2) Increased nitrogen in feces.

2. **Fats.** *Chemistry of Fats.* Human milk fats are esters of palmitic, stearic, and oleic acids with glycerin, the oleic acid ester being present in larger amount in human than in the cow's milk. Human milk fats are derived partly from body fat and partly from food fat. Carbohydrates also furnish ingredients for fat making; proteins do not.

Metabolism of Fats.

    1. Lipase from the gastric mucous membrane causes some splitting of fat.

    2. Fats are emulsified in small intestines.

    3. Live intestinal cells can change fatty acids to fats.

Resorption.

    1. Lymph-vessels.

    2. Blood-vessels.

Disposition.

    1. Subcutaneous tissue.

    2. Præperitoneal spaces.

    3. Liver.

    4. Burned with resulting end products.

        (1) Carbonic acid.   (2) Water.

In stools found normally as unresorbed portion of ingested fat in the form of

    1. Fat (neutral).   2. Lecithin.   3. Cholesterin.

    4. Fatty acids representing 1 to 10 per cent. of fat ingested.

    5. Alkali soaps.   6. Earthy alkali soaps.

In Urine. Fatty acids and glycerin are found in very small quantities, but we cannot say that these are from the fats ingested.

Nursing babies always have at least a small amount of fat in their stools. In contradistinction to proteins, the fats in the stools are in greater part only unresorbed fats, only a small amount being due to cell activity. (Proteins greater part).

Various percentages of fat ingredients found by Klotz in[1] examination of breast milk stools are as follows:

Neutral fat .... 29.5 per cent.
Fatty acids .... 10.7 "   · "
Combined fatty acids .... 58.8 "   "   (18.3 Ca and Mg.)

While under normal conditions 18.3 per cent. of the fats in the stools exist as non absorbable fat soaps in the so-called fat soap stools, they will approximate 50 per cent. of calcium and magnesium soap.

Fat in the Gastro-intestinal Tract and its Relation to Metabolism. Unlike proteins we can nourish the individual without fats, as carbohydrates can replace them. If too long continued, the organism changes, however, in its chemistry through increased absorption of salts and water.

3. **Carbohydrates.** Milk sugar formed by the mammary glands from material circulating in the blood is a disaccharide (glucose and galactose).

Chemistry of Carbohydrates.
 1. Monosaccharides.
   (1) Glucose (dextrose, grape sugar).
   (2) Lævulose (fruit sugar).
     They ferment and are reducible. (1) Has a right and (2) left polarization.
 2. Disaccharides.
   (1) Lactose—glucose and galactose.
   (2) Maltose—glucose and glucose.
   (3) Saccharose—glucose and lævulose.
     (1) and (2) are reducible, (3) is not.
 3. Polysaccharides (three or more sugar molecules).
   (1) Flour.
   (2) Dextrin.
   (3) Cellulose.

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[1] Langstein-Meyer: Wiesbaden, Verlag von J. F. Bergmann. Third edition, p. 16.

Metabolism of Carbohydrates. Monosaccharides are without further change absorbed in the small intestine or fermented.

Disaccharides are first reduced to monosaccharides by the intestinal ferments (every disaccharide having its specific ferment) before they can be absorbed. (This is not entirely true of maltose).

Polysaccharides are first acted upon by ptyaline in the saliva; this is continued in the stomach until the stomach content becomes acid, and then by enzymes of intestines and pancreas they are converted to monosaccharides.

After absorption into the blood, the carbohydrates serve the following purposes:

1. Used for energy.
2. Synthetically inverted into glycogen.
3. Fat foundation (probably).

Body cells can oxidize only monosaccharides (maltose excepted).

Interesting is the storing up of glycogen by the liver and muscles so that the sugar in the blood can be kept constantly at about 0.1 per cent.

Glycogen is most easily made from glucose and lævulose; less so from galactose, maltose and starch; least easily from cane and milk sugar.[1] [2]

Fat is formed from sugar by the subcutaneous cells, which are especially adapted to this function.[2]

Sugar is oxidized to carbon dioxide and water, which can be measured by the respiratory metabolism. Normally, sugar is absorbed from the small intestines, and is not found in the feces.

Sugar appears in the urine when the capacity for assimilation is passed, thereby producing an alimentary glycosuria. This is most easily accomplished in the

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[1] Otto von Furth: Physiological and Pathological Chemistry of Metabolism. J. B. Lippincott Company. p. 227-230.

[2] Langstein and Meyer: J. F. Bergmann, Wiesbaden, 1914, Third Edition, p. 16.

following order: lactose, galactose, laevulose, glucose. The cane sugar limit is about the same as milk sugar, while that of malt sugar is 7.7 grams per kilogram body weight. The assimilation limit for sugars is much greater in infants than in adults. An infant may develop mellituria when milk sugar exceeds 3.1 to 3.6 grams per kilogram body weight; in the adult at over 1 gram per kilogram. The height of the assimilation limit in itself shows that the infant's organism is adapted to a higher carbohydrate metabolism than that of the adult.

Carbohydrates in the Tissues. The newborn has a glycogen reserve which helps to sustain it until the appearance of the mother's milk.

Carbohydrates can, in part at least, replace proteins and fats. They cause a rapid increase in weight (very rapid at first), being deposited in the tissues, as glycogen, which latter can absorb two to three times its weight of water.

The relation of fats to carbohydrates is as follows:

The more carbohydrates present, the greater is the tendency on the part of the system to build up body fats. As to oxidation of fats, "They are burned up in the fire of carbohydrates" (Naunyns).

The complete burning of fats into carbon dioxide and water takes place only when the carbohydrate metabolism is normal; otherwise we get as mid-products the acetone bodies (acetone, aceto-acetic acid, oxybutyric acid, etc.).

They occur most frequently in infancy and childhood following periods of underfeeding or starvation. (Important in infants' disease, as seen during weaning, anorexia, continued fevers, intoxication, etc.)

Acetone bodies can also be formed from protein molecules. This occurs in starvation and in excessive meat and fat diets (deficiency of carbohydrates in the latter).

Weight becomes stationary or a loss results when carbohydrates are excluded or insufficient in the diet. Tem-

perature falls, and does not rise to normal until they are replaced.

4. **Salts.** *Chemistry of Salts.* Salts added to water are relatively split into their "ions"—that is, into either electrically positive or negative bodies. A solution of sodium chloride is a solution in which the NaCl molecule is intact, but the Na (kation) is electro-positive; the Cl (anion) is electrically negative.

Mature human milk contains 0.2 Gm. ash in 100 mils. Cow's milk 0.75 Gm. ash in 100 mils. Some exists as inorganic salts, others as important organic compounds.

I. *Kations* (or *cations*).

    1. Calcium.

        (1) Human 0.458 Gm. per 1000 mils, cow's 1.72 Gm. per 1000 mils, about 1 : 4.

        (2) Excretion is almost entirely through intestines, some from unabsorbed food remnants, and the rest by tissue metabolism.

    2. Magnesium.

        (1) Human 0.074 Gm. per 1000 mils, cow's 0.2 Gm. per 1000 mils.

        (2) Its metabolism is very closely related to the calcium.

    3. Sodium.   4. Potassium.

        (1) Human milk 0.132 Gm. Na_2O, cow's 0.465 Gm. Na_2O per 1000 mils, 1 : 3.

        (2) Human milk 0.609 Gm. K_2O, cow's 1.885 Gm. K_2O per 1000 mils, 1 : 3.

        (3) Excretion mostly through kidneys and stools.

    5. Iron.

        Human milk 0.0017 Gm. cow's 0.0007 Gm. per 1000 mils. These figures show considerable variation according to different authors. ˈExcreted mainly through the bowels.

II. *Anions.*

1. Chlorine.

Human 0.358 Gm., cow's 0.82 Gm. per 1000 mils, 1 : 3.

(1) Absorption: 90 to 100 per cent. through the intestine.

(2) Excretion: mostly through kidneys.

(3) About 0.5 per cent. retained by the system.

2. Phosphorus is contained in the milk in the following forms:

(1) Inorganic (calcium phosphate).

(2) Organic (casein, nuclein, lecithin, etc.).

(3) Total in human 0.345 to 0.418 Gm., in cow's 2.437 Gm. per 1000 mils, 1 : 9.

(4) Organic in human 43.3 per cent., and cow's 46 per cent., 1 : 1.

(5) The retention is higher in artificially fed than those fed on human milk.

Relation of Salts to Metabolism. The salts are necessary in digestion and in every step of metabolism from absorption to excretion and secretion. The rôle of these salts in both normal and pathological conditions has been given constantly increasing importance in the last few years.

Metabolism of Salts in Infants. In the gastro-intestinal tract the foods and salts are constantly changing action.

A casein product and calcium combine in the stomach to form calcium paracasein.

Fatty acids and alkalies and earthy alkalies in the intestines form soaps.

Casein increases excretion of salt in the intestine (moderate).

Fat increases excretion of salts in the intestines markedly, (especially Ca, Na, K). At the same time the phosphorus excretion decreases as the calcium phosphates are changed to calcium soaps by combination of calcium

with fatty acids, and the free phosphoric acid unites with sodium and potassium to form easily absorbed salts.

Salts are excreted in the urine and stools. The stools are the main channel of excretion of calcium, magnesium, and iron. Whether these are formed from the tissues or unabsorbed food is difficult to decide. The difference in percentages in human and cow's milk is equalized by the body using only what is necessary to its life and growth and not attempting to use it all.

Functions of Salts.

(1) They furnish building material for new cells. (Rachitis due to lack of absorption.)

(2) They are necessary to nerve excitability, muscle contraction, and many other vital functions.

(3) Addition of calcium and potassium to normal salt solutions counteracts their poisonous effects.

(4) Life is incompatible with withdrawal of minerals or even one ion.

(5) Life does not so much depend upon the ion as on its chemical combination. Therefore ash alone will not supply the needs.

(6) Infants need minerals for growth, as well as for life. Different tissues require different amounts and different salts.

(7) Weight drops with withdrawal of salts, even if other ingredients are constant, due to loss of water. Sodium salts are most important in water retention, calcium salts are least.

(8) Temperature falls, when salts are withdrawn (sodium).

(9) Phagocytosis is increased by calcium salts. Of value in infection.

5. **Water.** Infants need 105 Gm. of water, and adults 40 Gm. of water, per Kg.

Metabolism of Water. Intake is in the food. The outgo from the kidneys, bowels, lungs, and skin.

Water when ingested quickly passes through the stomach to be absorbed by the intestines. The water content of the organism varies with age and food. In the adult 58 per cent. of body is water, and in the newborn infant's body 66 to 69 per cent. is water. Sodium salts have the greatest facility for water retention.

Of the anions, Cl is the most marked in causing water retention.

Excretion of water takes place as follows: kidneys 59 per cent., skin and lungs 33 per cent., intestines 6 per cent. One to 2 per cent. of the water intake is retained.

Relation of Water to Metabolism. Approximately two-thirds of the infant's body is water. All cells need it; it is necessary to different combinations and reactions. In general, it is necessary for young infants on artificial feeding to receive about 140 to 150 mils (4 to 5 ounces) per kilogram (2 pounds) body weight every twenty-four hours. It carries nutritious material in the blood, lymph, cells, etc., and also the material for anabolism and katabolic products. It is also necessary to the function of the lungs and of the skin. Immunity to infection is to a large extent dependent on the water content of the body.

6. **Lipoids.** Proteins and lipoids form the principal component parts of all living cells. The lipoids are a group of organic nitrogenous substances comprising the phosphatids, cerebrosids and cholesterin. The phosphatids contain phosphorus, an organic base, and a fatty acid radicle in their molecule. The members of this series are *lecithin,* cephalin and cuorin. They are widely distributed in both animal and vegetable cells, but are especially abundant in the yolk of eggs, fish roe, brain tissue, yeast, blood and bile. They are also found to a lesser extent in cereal grains, legumes and beet root. The cerebrosids are isolated almost entirely from brain and nerve tissue. These do not contain phosphorus and yield galactose upon hydrolysis with dilute mineral acids. *Cholesterin* is an unsaturated secondary alcohol, is uni-

versally present in animal and vegetable tissue, and is most abundant in bile, yolk of eggs, nerve tissue and wool fat, and found abundantly in wheat, barley, beans, peas, lentils, carrots, peanuts and beets.

Lecithin. Lecithin is the fatty acid ester of the glycerophosphates (glycerin phosphoric acid), Human milk, 0.499 Gm. per 1000 Gm.; cow's, 0.63 Gm. per 1000 Gm.

Cholesterin. Human milk, 0.25 to 0.38 Gm. per 1000 Gm. Mainly excreted by the intestines.

This is of interest when we consider that fat-free milk contains but little lipoids.

7. **Vitamines.** The term is used to cover a group of substances the chemical nature of which has as yet not been determined. These compounds are absolutely essential in food, in order to maintain the weight of the body and produce growth. The lack of sufficient vitamines causes deficiency diseases, so named because they are due to a lack of something in the diet. It is unknown whether they exert their action directly on the tissues or indirectly, as has been suggested, through a hormone action. They cannot be produced by the animal organisms or are produced in such limited amounts that they are insufficient to meet the body needs. For this reason we are dependent upon the supply contained in the diet for these essential factors.

Lower forms of animal life, such as yeast cells, seem able to elaborate vitamines, and plant cells possess this faculty to a high degree. It is also impossible for the body to store them to any extent. Therefore it is necessary that the food contain a constant supply.

Tentatively they are grouped according to their solubility in fat and water and it may be hoped that a more scientific terminology will soon be applied to them when their chemical nature is better understood. At present they are described:

1. Fat-soluble A vitamine (growth promoting).

2. Water-soluble B vitamine (growth promoting and anti-neuritic).

3. Water-soluble C vitamine (anti-scorbutic).

The *Fat-soluble A vitamine* is thirty times as soluble in fat as in water. It is found in cod-liver oil, egg-yolk, butter, cream and milk. Because of the greater solubility in fats it is about equally distributed between the cream and the fat-free portion of the milk. It is contained in beef and mutton fat but little or none is found in lard and the commercial vegetable oils. It is found in considerable quantities in the heart, kidneys, liver and the glandular organs. The leaves of plants are rich in it, while the seeds and root vegetables contain less.

The *Water-soluble B vitamine* is one of the essentials in the promotion of growth. It is found in yeast, fruit juices, vegetables and grain embryos. The leafy vegetables and those growing above the ground, such as tomatoes and celery contain it in larger proportions than the root vegetables, such as potatoes, carrots, and turnips. It is also present in milk and egg-yolk. When cereals are very highly milled in order to obtain a very fine white flour, a large part of the vitamines may be removed. Vitamines are also lost when rice is polished in order to remove the outer layers, which contain most of the vitamines.

The *Water-soluble C vitamine* is known as anti-scorbutic vitamine. It is found in oranges, grapefruit, lemons and other citrus fruits (these contain both B and C), and in green vegetables such as tomatoes, spinach, lettuce and cabbage, and in eggs and raw milk. It is present in actively living cells, so that in general those vegetable tissues which contain relatively large numbers of actively respiring cells (leafy vegetables), are richer in anti-scorbutic power than are the roots or tubers. This generalization is not without exception. Different vegetables and fruits vary greatly in their anti-scorbutic potency. They differ widely also in the extent to which their anti-

NFANT FEEDING.

ll deteriorate under certain physical
ditions (drying, alkalinizing, etc.).
atement it is apparent that the anti-
f foodstuffs varies directly with the

ts on vitamines see Nutritional Dis-
Insufficient Vitamines, page 215, and

uff	Fat-soluble A or Anti-rachitic Factor	Water-Soluble B or Anti-neuritic (Antiberiberi) Factor	Antiscorbutic Factor
....	+ + +		
....	+ +		
....	+ + +		
....	+ +		
....	+		
tc. ...	+ +		
from			
....	Value in proportion to amount of animal fat contained		
....	+		
mutton,			
....	+	+	‡
....	‡ +	‡ +	
....	+ +	+	
....	+ +	+	
....	‡	‡ +	
....	+	+ +	
....	....	very slight, if any	
herring,			
....	+ +	very slight, if any	
....	+	+ +	
....	?	very slight	
raw	+ +	+	+
....		‡	+
....	less than + +	+	less than +
....	undetermined	+	less than +
weetened	‡	+	less than +
....			
....	‡ ‡	+ + +	?
....	+ +	+ + +	?
e, whole			
....	‡ +	‡ + +	

Classes of Foodstuff	Fat-soluble A or Anti-rachitic Factor	Water-Soluble B or Anti-neuritic (Anti-beriberi) Factor	Antiscorbutic Factor
Cereals, Pulses, etc.: (continued).			
Wheat, maize, bran	....	+ +	
Linseed, millet	+ +	+ +	
Dried peas, lentils, etc.....	....	+ +	
Soy beans, haricot beans ..	+	+ +	
Germinated pulses or cereals	+	+ +	+ +
Vegetables and Fruits:			
Cabbage, fresh (raw)	+ +	+	+ + +
Cabbage, fresh (cooked)	....	+	
Cabbage, dried	+	+	very slight
Cabbage, canned	....	...	very slight
Swede (rutabaga) raw expressed juice	.	....	+ + +
Lettuce	+ +	+	
Spinach (dried)	+ +	+	
Carrots, fresh raw	+	+	+
Carrots, dried	very slight		
Beetroot, raw, expressed juice	.. ..	....	+
Potatoes, raw	+	+	+
Potatoes, cooked	....	....	
Beans, fresh, scarlet runners, raw	....	....	+ +
Onions, cooked	....	....	+ at least
Lemon juice, fresh	....	....	+ + +
Lemon juice, preserved	....	....	+ +
Lime juice, fresh	....	....	+ +
Lime juice, preserved ..	....	....	very slight
Orange juice, fresh	....	....	+ + +
Raspberries	....	....	+ +
Apples	....	....	+
Bananas	+	+	very slight
Tomatoes (canned)	....	....	+ +
Nuts	+	+ +	
Miscellaneous:			
Yeast, dried	....	+ + +	
Yeast, extract and autolysed	?	+ + +	
Malt extract	....	+ in some specimens	

None of the three factors were found in:

Lard.

Olive, cottonseed, coconut or linseed oils.

Coco butter.

Hardened fats, animal or vegetable in origin.

Margarin from vegetable fats or lard.

Cheese from skim milk.

Polished rice, white wheaten flour, pure cornflour, etc.

Custard powders, egg substitutes, prepared from cereal products.

Peaflour (kilned).

Meat extract.

Beer

3. Milk Digestion.

1. In the Mouth. In the mouth milk is mixed with saliva, each 100 mils of milk averaging about 5 mils of saliva (Tobler). The secretion of saliva is stimulated mainly by the act of sucking, but also in part by appetite (psychic reflex). Ptyalin begins its action on the carbohydrates of the milk. Saliva may also cause coagulation.

2. In the Stomach. In the stomach the milk is curdled, casein being precipitated by rennin. Human milk coagulates less rapidly and less completely than cow's milk. Therefore in the latter the curds and the whey are more quickly separated.

Proteins are changed to albumoses and peptones by pepsin, and thus they are prepared for further digestion in the intestine. Albuminous digestive products stimulate gastric secretion.

Of *fats* 25 per cent. are changed to fatty acids and glycerin by lipase and action of bacteria. Fats at first retard, and later increase, the gastric secretion.

Action of ptyalin on *Carbohydrates* is continued for a time in the fundic end of the stomach.

Absorption in the stomach is as follows: (1) salts and sugars, (2) proteins (small amounts), (3) water (none), (4) fats (none).

Shortly after beginning of the nursing some of the whey content of the food begins to leave the stomach. This is more especially true if the ferments are active. The time also varies with the quality of the meals. Human milk leaves the stomach in about one and one-half to two hours after ingestion, and cow's milk in about three hours after ingestion. Two factors have an important bearing on this point: (1) the quantity of the fat, which delays the passage of the food through the pylorus, (2) the size of the curds, the large curds of the cow's milk delaying emptying of the stomach.

As previously stated, whey quickly passes out of the stomach, and remaining curd is digested at its surface, and thus passes over. Solid masses may pass through. After each passage of food the pylorus again closes. The rapidity of emptying the stomach depends on the action of the pylorus, and this in turn on the chemical composition of the food. Fats and albumins remain long in the stomach, sugars and salts passing through more rapidly.

3. **In the Small Intestines.** The action of the gastric digestion on the proteins is supplemented by trypsin from the pancreas, and the erepsin of the succus entericus. End products of the protein digestion are amino-acids. Carbohydrates are split into monosaccharides in the small intestines and are absorbed there. Fats which have been split into fatty acids and glycerin are emulsified and absorbed. Absorption of all digested food is almost complete in small intestines. It may be stated that intestinal or pancreatic digestion is far more important than gastric digestion in the infant.

4. **In the Large Intestines.** Absorption of water and excretion of salts are the chief functions of the large intestines in the digestive process.

5. **Feces and Urine.** *Feces* is composed of food remnants, products of secretory activity of the intestines, desquamated mucosa of the intestines, and bacteria. Composition of feces depends to a certain extent upon the nature of the food ingested. Foods rich in proteins (skim milk, albumin milk, etc.) cause increased intestinal secretion, with resulting alkaline reaction, which favors putrefaction and furnishes conditions favorable for development of fat soap stools. Excess of carbohydrates with acid fermentation gives another picture. Putrefaction and fermentation work antagonistically on the reaction of the stool. There is a balance between the acids derived from fat and sugars by bacterial action and the alkaline intestinal secretion.

Proteins in the stool (giving biuret and Millon's tests) are in greater part not derived from food proteins, but they are due to intestinal secretions, desquamated epithelial cells of the intestines, and to the bodies of bacteria. This is especially true of breast-fed infants. The normal infant stool contains no unchanged casein.

Fat has an important influence upon the formation of the stool. On feeding with human milk poor in fat the stools are small, containing small quantities of solids and some mucus. On feeding with human milk which is rich in fat, normal stools are produced. Microscopically fat is always evident in stools, and is derived partly from food, and in small quantities from the secretion of intestinal juices. Fatty acids and fat soaps are constantly found.

Salt excretion is an important function of the large intestine. In the breast fed, ash content of dry stool is 10 per cent., bottle fed 40 per cent. Insoluble calcium salts harden the feces.

The following are some tests on constituents of feces:

1. Fat soap easily seen as fatty acid crystals (needles) by heating with acetic acid on the cover glass and allowing to cool.

2. Carbofuchsin in weak solution stains as follows: Neutral fat: no stain. Soaps: faint rose color. Fatty acids: red.

3. Sudan III stains as follows: Neutral fat: orange red. Soaps: crystals do not stain. Fatty acids: stain red or crystals, orange red.

4. Sugar is not demonstrable in any quantity as such, but the character of the fat soap stool seen in milk feeding without sugar is changed to a softer, smaller, and normal color by adding sugar.

5. Starch is demonstrable by iodine test microscopically, but care must be exercised in the interpretation of the test, as the starch may be derived from baby powders.

The color of the stool is due to bile coloring matter derivatives: bilirubin and its reduction products, urobilin

and urobilinogen. The less the bile pigments are reduced, the more colored the stools. By marked reduction to urobilinogen, the color becomes almost white. The more milk and cream, *i.e.*, fat, in the diet, the paler the feces. The so-called soap stool is due to excess of fat and over-feeding with milk or cream with insufficient sugar, and is a firm grayish, putty-like stool.

Thin watery stools must always be taken seriously. However, the same cannot be always said of green, curdy stools, which are not infrequently seen in thriving breast-fed infants. These curds are almost invariably due to fatty acids and soaps.

Normal stools of breast-fed infants are homogeneous, salve-like, ochre-yellow color, acid, and of sour odor. Microscopically may be seen detritus masses, bacteria, few neutral fat corpuscles, and fatty acid crystals.

Normal stools of bottle-fed infants vary with the diet. One can frequently tell the diet by the appearance of the stool. On milk diet: less frequent, usually 1 or 2 daily, firmer and drier, usually pale yellow, alkaline and of foul odor. Constipation is the rule in babies receiving large quantities of milk with a moderate amount of carbohydrates. Sugars have a laxative tendency (fermentation). Excess of brown color may be caused by excesses of malt sugar. Starches, if well taken, tend to constipate, in large amounts they tend toward an acid reaction and an aromatic odor.

Starvation or hunger stool is seen on a very limited diet, as minimum amounts of milk, tea, cereal water. The stool has a dark, greenish-brown color, is soft, and composed in great part of mucus, and appears semi-transparent. This mucus may lead to further starvation through mistaken interpretation of its meaning, and result disastrously.

In the past it was taught that a study of the stools gave one definite information for the differential diagnosis of the gastro-intestinal diseases, but experience has taught us

that conclusions are of value only when based upon stool examinations in conjunction with a careful study of the diet, and clinical examination of the infant.

Urine. A normal infant urinates ten to fifteen times daily, and the urine passed represents 60 to 70 per cent. of the fluids taken as food and drink. It is acid in reaction, and should be free from albumin. However, albumin frequently is present in the simple nutritional disturbances, and almost constantly in severe acute illnesses. Temporary presence of albumin in the urine of the newborn may be considered physiological, as well as the uric acid during the very early stage. Great decreases, even to anuria, are common with the intestinal disturbances.

CHAPTER IV.

BACTERIA OF THE DIGESTIVE TRACT OF THE INFANT.*

1. The Newborn.

FOR about one day the meconium passed by the newborn baby is sterile. During this time, however, the bacteria begin to invade the digestive canal of the infant through the mouth and through the anus. The initial intestinal flora which thus develops is subject to marked differences, the number and nature of the bacteria depending chiefly upon the surroundings of the infant, and exhibits no characteristic constant findings.

This period is followed by gradual transition in the nature and in the number of the intestinal bacteria, until about the third day after birth characteristic intestinal flora become established, consisting chiefly of Bacillus bifidus (in the nursing infant) and Bacillus coli (in the artificially fed infant), and, besides these, Bacillus acidophilus, Micrococcus ovalis, Bacillus lactis aërogenes and others.

2. The Nursing Infant.

The principal portal of entry of the intestinal bacteria is the mouth. There is no doubt that a great variety of organisms may from time to time enter this atrium, including not only the ordinary organisms of the nursling's environments, but pathogenic bacteria as well. A majority of these pass to the stomach, and they may pass to the intestinal tract.

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* In the elaboration of this chapter free use has been made of A. I. Kendall's Bacteriology, Lea & Febiger, Philadelphia and New York, 1916.

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. The flora of the mouth and of the stomach are not well known, but they appear to be of relatively slight importance as a rule.

The duodenal flora in health is composed chiefly of coccal forms of the Micrococcus ovalis type. Bacillus coli and other members of the colon group are most numerous at the ileocecal valve and the cecum, and Bacillus bifidus or similar organisms dominate the large intestines from this level to the sigmoid flexure. The remainder of the large intestines to the rectum is somewhat sparsely populated with living bacteria, partly because the fecal mass is relatively desiccated by the absorption of water, partly because of the accumulation of waste products of bacterial activity—principally acids resulting from fermentation of lactose, formed higher up in the tract—which inhibit the development of bacteria in the lower levels.

Bacillus bifidus (Gram positive, blue stain) predominates in the intestinal flora of the breast-fed infant, being acid tolerant and finding favorable conditions for its growth and development, since in digestion of mother's milk lactic acid production from lactose is so great as to inhibit the growth of the Bacillus coli and Bacillus lactis aërogenes in the lower end of the ileum, while the highly acid medium favors the growth of the Bacillus bifidus communis and the acidophile bacteria. Coccal forms and lactose fermenting organisms are present, but scanty; spore bearers are rare.

3. Artificially Fed Infants.

Escherich directed attention to the striking dissimilarity between the intestinal flora of the breast fed and the artificially fed infant. Culturally, morphologically, and chemically the former is more uniform than the latter. The most distinctive features of the dejecta of the artificially fed infants are: the relative increase of Gram-negative bacteria of the coli-aërogenes type, and of coccal

forms of the Micrococcus ovalis type, together with a diminution of Bacillus bifidus. Bacillus acidophilus is relatively more numerous, as a rule, in the artificially fed infant than in the nursling. Proteolytic bacteria of several types are also of frequent occurrence, but they are not commonly found in the dejecta of the normal nursling. These organisms are frequently spore-forming bacilli, of which two principal groups are recognized —members of the aërobic group, of which Bacillus mesentericus is a prominent type, and anaërobic bacteria, of which Bacillus aërogenes capsulatus is most widely known; it frequently occurs in small numbers in the feces of artificially fed infants. The reaction of normal feces of artificially fed babies is usually alkaline; culturally and chemically, the evidence of intestinal proteolysis of bacterial causation is more marked in these infants than in normal nurslings.

The general distribution of types of bacteria at the different levels of the intestinal tract is similar to that observed in normal nurslings. The principal differences are found in the cecum and large intestine, where the obligately fermentative bacteria of the bifidus type are replaced to a considerable degree by an extension of the habitat of the Bacillus coli, of Bacillus acidophilus, and the appearance of moderate numbers of proteolytic bacteria, both aërobic and anaërobic; many of the latter are sporogenic.

The characteristic feature of the normal adult fecal flora as compared with the infantile nursling flora is the very heterogeneous variety of types of bacteria in the former, in sharp contrast to the homogeneity of types of bacteria in the latter.

4. Significance of the Intestinal Bacteria.

The striking differences in morphology, chemistry, and in cultural characters between the intestinal floras characteristic respectively of nurslings, artificially fed infants

and adults suggest at once that nutritional stimuli may be an important factor in determining the dominance of type of bacteria. It is probable that the significance of the intestinal flora lies rather in its potential antagonism to alien bacteria, which certainly gain entrance to the alimentary canal from time to time, than in any specific participation in the normal digestive process of the host. The normal intestinal flora may be regarded as intestinal parasites, just as the various bacteria which occur commonly on the skin are regarded as cutaneous parasites. It is important to realize that the normal intestinal organisms, like the cutaneous organisms, are "opportunists," potentially capable of becoming invasive whenever the barriers which ordinarily suffice to limit their development to the lumen of the alimentary canal become impaired, giving rise to endogenous infections.

5. Influence of the Diet on the Intestinal Flora.

Intestinal flora vary greatly, the most important factor in determining its nature being the chemical composition of the food. Human milk gives essentially different flora from cow's milk. There are two groups of bacteria possessing an antagonistic action, those causing fermentation (saccharolytic), and those causing putrefaction (proteolytic). The representatives of the former are Bacillus lactis aërogenes and Bacillus bifidus, the latter being the most important organism in the stool of the breast-fed infants. The group exercising proteolytic activity is less clear. We know only that in the processes of putrefaction the bifidus flora is replaced by the coli group. Depending on the predominating group of bacteria, putrefaction or fermentation takes place, causing either firm or soft stools, this rather than the activity of the ferments determining the nature of the stools. The nature of the food and its chemical composition, therefore, determines the nature of the development and activity of the particular bacteria in the intestinal tract.

The human milk, rich in sugar and low in protein, leads to the flora of fermentation, while cow's milk, rich in protein and poor in sugar, to the flora of putrefaction. This phenomenon is nothing specific, but is due to individual components of the milk and their mixture.

Carbohydrates lead to the development of the fermentative organisms; the split products of carbohydrates are acetic, butyric, lactic and carbonic acids.

The nature of the dominant organisms which develop in diets rich in carbohydrates varies with the carbohydrate itself. Bacillus bifidus is more commonly predominant when lactose is the sugar fed, without an excess of protein. If maltose or dextrose is substituted for lactose under the same conditions, Bacillus acidophilus is very frequently the more prominent.

The fermentative action is increased by sodium and potassium salts as found in whey. (This latter probably in part explains the results obtained in feeding malt sugars together with potassium carbonate.)

Proteins favor the development of the organisms of putrefaction and lead to formation of indol, skatol, and amino-acids, these being the products of aromatic and fatty series. Gases are also formed by the latter action.

The nature of the protein influences the types of proteolytic bacteria to a very marked degree. In general, animal proteins other than casein appear to encourage somewhat more active proteolytic flora than vegetable proteins. The processes of putrefaction are favored by calcium salts.

The influence of *fat* in its relation to bacterial processes is not clear. It seems to be able to favor fermentation, if this be already present, and also to increase the intensity of the processes of putrefaction.

In breast feeding fermentation outweighs putrefaction. The question whether fermentation or putrefaction in the intestinal canal is desirable, must be answered *a priori* that the fermentative processes are physiological, since

breast feeding always leads to this. By this it must not be understood that the putrefaction in artificial feeding causes injury. Excessive intestinal fermentation in artificial feeding may be the forerunner of disaster, and is to be avoided (dyspepsia, intoxication).

Within certain limits, we are able to influence the bacterial processes in the intestinal tract in the normal infant, and thereby change the character of the feces. In a sick infant this is more difficult, and larger quantities of putrefacient food are necessary to overcome pathological fermentation.

6. Intestinal Bacteria in Their Relation to Gastrointestinal Disturbances.

There are many intestinal disturbances of unknown causation, presumably unrelated to bacterial activity. There is a second group of conditions in which bacteria may conceivably play a secondary part; in some of the latter abnormal physiological conditions in the alimentary canal may be justly regarded as the antecedent factors. The boundaries of these two groups are poorly circumscribed, and they merge through imperceptible or poorly defined limits into a third group of cases in which the activities of endogenous or exogenous bacteria in the alimentary canal may be the causative factor in morbid processes of the gastro-intestinal tract.

The symptomatology induced from the products arising from the *decomposition of proteins or protein derivatives* by the action of bacteria in the intestinal tract depends largely upon the organism or organisms concerned. It varies from the somewhat insidious, slowly progressing, so-called autointoxication, in which a marked increase of urinary ethereal sulphates may be a suggestive index, to the acute toxemias characteristic of bacillary dysentery, typhoid, paratyphoid or cholera. Of course, a variety of other bacteria than the few mentioned specifically may be concerned, either alone or in symbiosis.

Thus streptococci alone, and streptococci in association with dysentery bacilli, may be justly regarded as the etiological agents in their respective syndromes. The important factor, from the viewpoint of this discussion, is to realize that the formation of nitrogenous products from proteins or protein derivatives, which are being utilized by various types of intestinal bacteria for energy, may be injurious to the host.

The other prominent type of abnormal bacterial activity in the alimentary canal—the fermentative type—is of entirely different origin. The essential factor is either a *fermentation of carbohydrates*, with the formation of products abnormal for the intestine, or of excess of normal fermentative products. The factors leading to an overgrowth of these organisms in the intestinal tract appear to be an excess of carbohydrate and a lack of normal lactic-acid-forming bacteria.

It is unfortunate that practically none of the bacteria which incite intestinal disturbances or illnesses produce soluble toxins against which antitoxins can be prepared. Sera likewise have been unsatisfactory. There is little, therefore, that can be accomplished serologically with the present methods in the treatment of intestinal disturbances of bacterial causation. Attempts to permanently eliminate or destroy undesirable bacteria with cathartics and intestinal antiseptics have not been productive of results in the past, and prolonged starvation *per se* does not lead to intestinal sterility or to a significant reduction in the offending bacteria.

There are two ways, however, in which direct influence may be applied to bacteria in the intestinal tract: by substituting harmless types of organisms for abnormal types, and so varying the diet for space that the intestinal contents at the desired level shall contain nutritive substances that may be reasonably expected to shift the metabolism of the offending organism, and therefore radically change the character of the products of its metabolism.

Diseases Due to Proteolytic Activity of Bacteria.
There are a number of conditions of bacterial causation
in which available evidence points strongly to the forma-
tion of products arising from the metabolism of protein
or protein derivatives by specific organism as important
etiological factors in the morbid process. Thus, cholera,
bacillary dysentery, typhoid, paratyphoid, and many less
acute infections are associated definitely with the de-
velopment of these organisms within the body, and to
some degree at least, at the expense of the body tissues.

Available evidence points strongly to the view that
cholera vibrios, typhoid, dysentery and paratyphoid bacilli
and similar organisms produce their characteristic and
harmful effects when they are developing in media free
from utilizable carbohydrates; when utilizable carbohy-
'drates are added to these media, non-characteristic, harm-
less products are formed.

In the absence of any definite indication to the con-
trary, it would be logical to attempt to maintain a suffi-
cient concentration of carbohydrates within the intestinal
canal in these infections as a therapeutic measure.

The important effects to be accomplished by a liberal
carbohydrate diet in those infections where the decom-
position of proteins or protein derivatives by bacterial
activity leads to chronic or acute illness of intestinal
origin are: a change in the metabolism of the offending
organism resulting in the formation of lactic and other
acids in them in place of putrefactive products, and a
gradual replacement of the proteolytic and pathogenic
types by bacteria of the fermentative varieties.

*Diseases Due to Excessive Fermentation of Carbohy-
drates.* Another type of intestinal disturbances depends
upon an unusual or an excessive fermentation of carbo-
hydrates. This is frequently seen in young infants, in
many of whom we have a limited carbohydrate tolerance.
(See Nutritional Disturbances.)

PART II.

The Nursing.

CHAPTER I.

GENERAL CONSIDERATIONS.

WRITERS on this subject are very prone to state that the ability of the mother, particularly among the well-to-do, to fulfil this most important function is decreasing. This may have been a true statement fifteen or twenty years ago. At the present time, however, we are sure it is erroneous. The young mother of to-day is better able to nurse her offspring than was her sister fifteen or twenty years ago. We attribute this to the fact that the youth of the present day are more vigorous, more nearly normal individuals, than were those of an earlier date. Breast-milk during the first two or three weeks of the infant's life is produced under unfavorable conditions, which do not indicate the possibilities of the breast as a secreting organ. Early nursing, following as it does upon the stress of confinement, is not indicative of what may be possible later, when the customary life and daily habits are resumed. Repeatedly we have found a very high fat or a high protein, or both, entirely corrected after the first week or two, without interference. This condition at the time was considered sufficiently serious to warrant the discontinuance of nursing on the part of a weakly infant, while in a vigorous infant it would be entirely ignored. A neurotic mother makes the poorest possible milk-producer. Proportionate to the population, there are fewer neurasthenics among the young women to-day than there were twenty years ago, and

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there will be still fewer twenty years hence. At the present time the timid, retiring young woman of the neurasthenic type is not popular in her set.

Few functions with which we have to deal are so variable and uncertain as the production of breast milk. Breast milk is one of the most precious substances. It is invaluable, unless we can put value on human life. The most successful nursing age is between the twentieth and thirty-fifth year.

Some mothers will be able to carry on the nursing for only two months, others three, five, seven, or nine months. In our experience in both out-patient and in private practice it is extremely rare for the breast milk to be sufficient for the infant after the ninth month.

It should be remembered that besides the protein, fat, carbohydrate, salts and water content there are other bodies contained in human milk, which, even though not essential to the infant's life, are of inestimable value to it. These may be divided into two groups:

1. Immunizing bodies—antitoxins, alexins, etc.— which are contained in the mother's blood, and transmitted to the baby through her milk. They are of value in protecting the infant against infections.

2. Ferments: lipase, galactase, lactokinase, and diastase.

Examination of Human Milk. This is rarely of any practical value. The protein rarely causes trouble, and the sugar is usually constant (6 to 7 per cent.). The examination of milk is therefore usually restricted to a determination of the fat content by means of the lactometer. The richest milk, however, will usually agree with the baby, and it is apt to thrive equally well on a milk that shows a small amount of fat. In other words, the baby and not the lactometer is the only practical test. If the milk disagrees, it will be evident clinically. *No baby should ever be deprived of its mother's milk only*

because of the results of a clinical examination of the milk.

In making an examination of the mother's milk one must bear in mind that the first milk is very poor, the last very rich in fat, and that an average specimen can be obtained only by mixing the whole amount, or by combining the first and the last, or, better still, by taking only the middle portion after a few drams have been drawn off. This can be accomplished by allowing the infant to nurse for two minutes before expressing the sample.

Contraindications to Nursing. *Tuberculosis* when progressive or open is always a contraindication to nursing, because of the danger to the infant and the strain on the mother. With proper precautions, and where the breast is not diseased, and human milk is not obtainable from other sources, it may be well to tide a weak infant over its first weeks by expressing the milk from the mother's breast.

Syphilis of the mother, except in freedom from infection on the part of the infant, is not a contraindication. Lack of symptoms on the part of the mother in congenital syphilis is a very common occurrence; a Wassermann reaction on the mother's blood will quickly clear up any doubt.

Any *grave constitutional disease* in which there is an extraordinary drain on the resources of the body (diabetes, heart disease with disturbed compensation, nephritis, Basedow's disease, malignant neoplasms, epilepsy and psychoses) are contraindications to nursing.

Acute diseases should only in exceptional cases be considered as contraindications to nursing, and should include conditions in which there is danger of overburdening the mother and infections endangering the infant. (See p. 65 for further indications.)

CHAPTER II.

MATERNAL NURSING.

1. Nursing Axioms.

The following may be laid down as nursing axioms:

A diet similar to what the mother was accustomed to before the advent of motherhood can usually be taken.

There should be one bowel evacuation daily.

From three to four hours daily should be spent in the open air in exercise which does not fatigue.

At least eight hours out of every twenty-four should be given to sleep.

There should be absolute regularity in nursing and expression.

There should be no worry and no excitement.

The mother should be temperate in all things.

2. Hygiene of the Mother.

The Diet of the Mother. Many times, when consulted by nursing mothers because the nursing was unsuccessful or a partial failure, we have found that their diet had been restricted to an extreme degree. To put on a greatly restricted diet a robust young mother who has always eaten bountifully of a generous variety of foods is one of the best means of curtailing the quantity and lowering the quality of her milk supply. When asked to prescribe a diet, we tell such mothers to eat as they were accustomed to before the advent of pregnancy and motherhood. That this particular vegetable or that particular fruit should be forbidden on general principles is a fallacy. Food that the patient can digest without inconvenience is a safe food so far as the nursing is concerned, as may readily be determined in any given case. For certain individuals, however, a plain, more or

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less restricted diet is desirable. This must be enforced in the management of the wet-nurse (to be detailed later).

Nursing is a perfectly normal function, and a woman should be permitted to carry it out along the natural lines. Inasmuch as there are two lives to be provided for instead of one, more food, particularly of a liquid character, may be taken than the mother may be accustomed to. It is our custom to advise that milk be given freely. A glass of milk may be taken in the middle of the afternoon, and 8 ounces of milk with 8 ounces of oatmeal or cornmeal gruel at bedtime, if it does not disagree with the mother. Our only evidence that a food is disagreeing is the condition of the digestion. When any article of food disagrees with the mother, or if she is convinced that it disagrees, whether or not such be really the case, the food should be discontinued. In a general way, milk (one quart daily), eggs, meat, fish, poultry, cereals, fresh vegetables and fruits constitute a basis for selection. Although occasionally a nursing mother cannot take acid fruits, salads and aromatic vegetables, they may be tried and discarded, if they disturb the infant. Eggnogs, thin cereal gruels mixed with milk, cocoa and malted milk and similar drinks can often be taken to advantage between meals.

The Bowel Function. A very important and often neglected matter in relation to nursing is the condition of the bowels. There must be one free evacuation daily. For the treatment of constipation in nursing women we have used different methods in many cases. The dietetic treatment and plenty of recreation and exercise promise most. Manipulation of the diet should not be such as to interfere with the milk production. Three other methods are open to use: massage, local measures and drugs. Massage is available in comparatively few cases. Local measures consist in the use of enemas and suppositories. Every nursing woman under our care is instructed to use an enema at bedtime, if no evacuation of

the bowels has taken place during the previous twenty-four hours. For a laxative in such cases and in many others, a capsule of the following composition has served well:

R Extracti nucis vomicæ .... 0.015 Gm. (¼ gr.).
    Extracti cascaræ sagradæ .... 0.325 Gm. (v gr.).
    Sig.: To be taken at bedtime.

The amount of the cascara sagrada may be varied as the case may require. In not a few instances we have found it necessary to give 2 capsules a day in order to produce the desired result. Neither the nux vomica nor the cascara appears to have any appreciable effect on the child.

Air and Exercise. Outdoor life and exercise are not only as desirable here as they are under all other conditions, but to the nursing woman, with her added responsibility, they are doubly valuable. In order to get the best results, exercise or work should be so adjusted as not to reach the point of fatigue. The mother whose nights are disturbed should be given the benefit of a midday rest of an hour or two. It should be our duty, however, to explain to the mother and to other members of the family that an important element in satisfactory nursing is a tranquil mind.

Care of the Breasts. A well established routine should be instituted for the care of the breasts. To facilitate this a readily accessible tray with the necessary utensils should be provided. This should contain a glass-stoppered bottle with a saturated solution of boric acid, a jar of cotton pledgets on toothpicks, to be used as applicators for the boric acid, a graduated glass or beaker. The nipples should be thoroughly washed before and after nursing with a saturated solution of boric acid poured fresh from the bottle for each cleansing, and the surplus thrown away. The boric acid should be applied with the cotton pledgets. The fingers should not come

in contact with the nipples, if the child is to nurse directly at the breast. If the nipples are tender, they should be anointed with a sterile mixture of 5 per cent. tincture of benzoin in liquid vaseline.

All utensils, including the breast-pump, if one is in use, should be sterilized by boiling. In case of the breast-pump, the rubber bulb may be removed for this purpose. Where the milk is to be expressed by hand, the hands must be thoroughly disinfected by washing with soap and water, and rinsing with alcohol before manipulation of the breasts. Under all conditions soap and water should be freely accessible, and their use required before handling the breast or the infant.

3. Conditions Influencing the Breast Milk.

The advent of the first *menstruation* period particularly, and in some cases the beginning of every menstruation period, is attended with an attack of colic or indigestion in the child. Such attacks, however, rarely necessitate the discontinuance of the nursing even for a single day. Not infrequently the quantity of milk is somewhat lessened during menstruation, and this will result in the infant becoming fretful, due to insufficient quantity of the feeding. Under no circumstances should menstruation be considered an indication for weaning.

Factors influencing the *mental condition of the mother*, such as anger, fright, worry, shock, distress, sorrow, or the witnessing of an accident may affect the milk secretion sufficiently to cause no little discomfort to the child, and oftentimes the lessening of the flow for a day or two. At times, especially when the mother is under influence of shock or grief, it may be necessary to substitute artificial feeding for a few nursings during these periods, until the mother has again resumed her mental equilibrium, her breast being emptied by mechanical means in the meantime.

Drugs, alkaloids of opium, hyoscyamus, belladonna, and similar drugs, when given in large quantities, not infrequently pass into the milk, and should therefore never be administered in large quantities to the nursing mother. Belladonna may cause a decrease in milk secretion, and should be administered with caution during the period of lactation. Mercury, iodides and the newer salts of arsenic are also secreted in the milk, and may be used to advantage when a luetic mother is nursing a luetic infant.

4. The Nursing Proper.

Regularity in Nursing. The breast which is emptied at definite intervals invariably functionates better than does one which is not, not only as regards the quantity, but also the quality, of the milk, thus regular habits in breast-feeding are as essential to milk production as to its digestion and assimilation. *The baby should be wakened to be fed.*

The average mother will supply the needs of the individual meal with *one breast,* and the breasts should be alternated in successive feedings. Thorough emptying of the breast should be encouraged under all circumstances, as this is our best method for increasing the milk supply, and the baby is the only means at hand by which this can be accomplished. This should be encouraged in every instance. It is most readily thwarted by allowing a lazy baby to partially empty both breasts, and will soon lead to a diminished milk secretion. By this means the mother and the baby soon become adapted to one another, and it will be found that the desired effect is accomplished both where the milk supply is insufficient or, again, excessive. In the former instance complete emptying of the breasts increases the secretion, and, where excessive, incomplete emptying will soon result in a lessened supply.

Sometimes, however, it is advisable to give *both breasts* at each feeding, *i.e.,* under the following conditions: (1)

During the first few days, to stimulate secretion, and a little later to relieve the congested breasts; (2) to weak babies when there is an abundance of milk, and they are not strong enough to get the last milk that comes harder; (3) to overfed babies, where it is desirable to give them only the first and weakest milk, and to lessen the yield of the milk from the breast; (4) as the milk supplied by one breast fails to meet the needs of the infant, both breasts should be given at each nursing; the first breast should be thoroughly emptied before allowing the baby to take the second breast, and the next nursing started on the second breast given in the last feeding.

Number of Feedings in Twenty-four Hours. Four-hour intervals at start with six feedings in twenty-four hours, five feedings by the second to the fifth month, according to the individual needs of the child. Night nursing can often be discontinued by this time, and babies properly fed will go from 10 P.M. to 6 A.M. without anything but perhaps a drink of water.

Premature and delicate infants and infants with a tendency to vomit are exceptions, and must be fed smaller amounts at more frequent intervals.

Length of Nursing. As a rule a robust baby takes three-fourths of the milk obtained from a good breast in the first five minutes of a twenty-minute nursing. Fifteen to twenty minutes should be the limit for the nursing period.

The quantity received at individual nursings will vary greatly throughout the day. The early morning nursings will often yield twice the amount of the later nursings. Therefore it is necessary to ascertain the twenty-four hour quantity in order to estimate the total value of milk received.

When one breast does not meet the infant's demands both breasts should be given at each feeding, the normal nursing time of fifteen or twenty minutes being divided between the two breasts, either equally or by alternating

a long and short feeding period of fifteen and five minutes, so that each breast will receive a long nursing period at alternate feedings. Weak and lazy babies may require awakening during the nursing period to keep them at work. Very weak babies may require a longer period with short intervals in which they rest.

The Daily Total of Milk Required. Most young infants will satisfy their requirements for growth and development when receiving an average of two and one-half ounces (75 mils) of human milk per pound (½ kilo.) body weight, in twenty-four hours. Roughly this may be stated as one-sixth of the body weight in milk daily (50 calories per pound). Older infants will usually thrive on two ounces (60 mils) or 40 calories of breast milk per pound or one-eighth of their weight.

While infants of the same weight and age under the same conditions will require practically the same amounts to provide for growth and development, on the whole the fat baby will require less per pound than the thin one.

Water Requirements. When the infant is receiving one-sixth of its body weight in milk during the day, little, if any additional water is required. When the breast-milk does not meet this requirement additional water or other food must be administered to meet the required one-sixth of the body weight in fluids. During the first days of life, when the breast milk supply is insufficient, total fluids should be administered to meet the above needs. From one to three ounces of a 2 to 5 per cent. solution of cane or milk sugar which has been boiled, may be given to the infant at twenty-four hour intervals until the milk appears.

Before giving the water, the infant should be placed at the breast at each feeding. Even when milk is plentiful the administration of water two or three times daily from a nursing bottle accustoms the infant to taking the food in this way. An infant so trained will meet emer-

gencies of weaning more readily than one unaccustomed to bottle feeding.

Feeding During the First Days. During the first day of life, food may be withheld for twelve hours, the infant being kept in a warm crib. It usually soon falls asleep, and as a rule it should be awakened only to change diapers. As a rule the child does not evidence its initial sensation of hunger by crying until after its first half day of life, and even then in many cases it is difficult to obtain the co-operation of the infant in administering its food. During the second twelve hours the infant may be put to the breast two or three times in order to stimulate secretion and to teach it to nurse. During the second twenty-four hours the baby should be put to the breast at regular four-hour intervals. The sixth feeding may be omitted. By the third or fourth day the infant will usually receive most of its required food from the breast. If a night feeding is to be instituted it is well to waken the baby at the regular hour in order to cultivate regular habits, which are so essential to the mother's welfare.

The Total Nursing Period. Some mothers will be able to carry on the nursing for only two or three months, others as long as nine months. In out-patients as well as in private practice, it is exceptional to find a breast-milk supply which is sufficient for the infant after the ninth month. It is usually wise to allow one bottle feeding daily, by the end of the third or fourth month, in order to relieve the mother, and at the same time train the baby in bottle feeding.

CHAPTER III.

WET-NURSING.

1. The Wet-nurse: Her Selection and Her Baby.

The Problem. When there is a positive inability on the part of the mother to nurse her offspring, either through inadequate functioning on the part of the breast or systemic disease, we are confronted with the problem of securing human milk from another source, as notwithstanding the numerous isolated reports on successful raising of infants on artificial foods, the statistics of infants fed by artificial foods when compared with those of infants fed on human milk are so strikingly in favor of the latter that the obtaining of human milk must always be considered as an important issue.

How Obtained. In our experience, even in a large city, great difficulty has been met in obtaining a regular supply of wet-nurses. On several occasions various charitable and hospital societies have attempted to establish a wet-nurses' registry as a clearing-house for the several maternity and general hospitals of Chicago. These attempts have not been successful for two reasons: (1) because of the irregularity in the demand, and (2) because of the lack of co-operation on the part of the various institutions caring for this class of cases.

The Nationality of the Wet-nurse is of considerable significance where the supply allows of a selection. The phlegmatic temperaments as seen in women of Northern and Central Europe of Teutonic and Slavic descent, offer the ideal material, while other nationalities, such as Italians, and the Southern negroes when removed from their home environment to a Northern climate, with the consequent change in diet, secrete a milk poor in quality.

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However, even the latter in an emergency should not be neglected.

Requirements of a Good Wet-nurse. 1. She should be in good health, and, especially, free from all contagious and infectious diseases, and also from local diseases of any kind, such as those involving the nose, throat, skin, etc.

2. Her mammary glands should be of such quality that she can secrete sufficient milk of good quality, and the nipples sufficiently developed to allow of nursing, or proper expression of the milk.

3. Whenever possible, her age should be not less than 18 and not more than 35 years.

4. The age of her baby, as compared with that of the baby she is to nurse, is a matter of indifference in most instances. However, the first weeks, or if possible the first two months, of lactation should be avoided, because of the presence of colostrum and the rapidly changing quality of the breast milk, which not infrequently causes serious gastric and intestinal disturbances in very susceptible infants, as evidenced by vomiting, colic and diarrhea. Multiparity may be considered an asset, if the nurse has demonstrated her ability to care for and feed previous cases. A multipara is also less likely to be affected by her new surroundings, especially if this be a private home. When the wet-nurse has more or less direct charge of the infant, one who has been nursing her own or other infants will be more likely to meet the technical difficulties in the care of her charge.

Examination of the Wet-nurse. The examination of the wet-nurse should always be made in a systematic manner to insure against overlooking important things.

First, a careful history should be taken as to the number of her children, miscarriages, and the presence of constitutional diseases in her family.

Second, she should be thoroughly examined, all parts of the body being exposed, and the examination should

include the skin and hairy parts of the body for the presence of skin lesions and parasites, as well as for old luetic scars. The organs of the chest and abdomen should be subjected to careful examination.

Third, the breasts should be examined.

Fourth, the genitalia, including the cervix and the urethra, and in all cases a cervical (and where suspicious, a urethral) smear should be taken and examined for gonococci. As a single smear is often misleading, in cases of the slightest suspicion, where a girl baby is to be nursed, the examination of the cervical and urethral smears should be repeated.

Fifth, an examination and search should be made for chronic infections, especially for syphilis. A Wassermann test should be made in every case, and reported upon before she is allowed to supply milk, as it is well known that a syphilitic mother in a very great number of cases shows no clinical evidence of syphilis. The mouth and pharynx, neck, anus and genitalia, entire skin and lymphatic glands should also be examined for evidence of syphilitic lesions.

Tuberculosis. The lungs, glands, and osseous system should be examined, and a careful history as to susceptibility to colds and to recurring bronchitis elicited.

Sixth. Acute infections. She should be questioned as to exposure to contagious disease, and she should be examined for evidence of acute infections of the nose, throat, and ears.

Seventh. Her teeth should be examined and defects and pyorrhea corrected, if necessary, at the expense of the family.

Eighth. The urine should be examined (1) for evidence of nephritis, (2) for evidence of diabetes. It should, however, be remembered that a positive reaction for sugar should not be overestimated, unless the sugar is proven to be dextrose, as very commonly in our experience during the early weeks of lactation a lactosuria

is present. The kind of sugar can easily be determined by the phenylhydrazine test, followed by a microscopical examination of the crystals.

Ninth. Nervous and psychic disturbances, such as epilepsy, insanity, hysteria, should be cause for rejection of the nurse.

Tenth. Her child should be examined for evidence of syphilis. Possibly one of the best arguments for the non-employment of a wet-nurse during the first two months of her lactation is the possibility of a latent syphilis. Where there is the slightest doubt, a Wassermann reaction should be made on the infant. The general condition of the child gives us the best evidence both as to the quantity and to the quality of the maternal milk. Unless the source of the nurse be known, it is well to be certain that she is nursing her own baby. In case of its death or its absence, every effort should be made to obtain its condition at birth and its later development.

So far as possible she should not be subjected to annoying questioning on the part of the family, which is entirely unnecessary, if she has been properly examined by the physician. It has been our experience that such unnecessary questioning has led to nervousness, and not infrequently has caused her to decline the position, at a time when she was most needed.

Her Place in the Household. She should be treated neither as a guest nor as a menial, but so far as possible should be graded according to her previous station in life. There is a grave danger of mental depression on the part of a woman, well-born and sensitive, who, through misfortune or necessity, is forced to seek this means of employment, and also of an exaggerated estimate of self-importance on the part of a woman but little accustomed to the luxuries of life upon her entrance into the home of employment, particularly if attentions are paid to her. As has been previously stated, all instructions

and demands should be made by the person best qualified in the individual case. A divided responsibility will always lead to future complications.

Her quarters should be well located; their ventilation should be supervised, and she should be held responsible for their general cleanliness. The wet-nurse's baby should always be kept in the room with her, so that she may feel the full responsibility for its health and care.

The Quantity of Milk to be Expected from a Good Wet-nurse. The quantity and quality of milk supplied must vary greatly with the glandular development of the individual wet-nurse, the state of her health, and the factors quoted elsewhere which would affect it temporarily. The amount and variety of stimulation applied to the breasts, of which the direct nursing by a full-term infant is the most valuable (at least for the purpose of stripping the breasts), must be given due consideration. In view of the many emergencies and influencing factors, no absolute standard for quantity and quality can be set for general rule.

A wet-nurse who does not secrete sufficient milk during the first few days in her new employment should not be discharged until every effort has been made to improve her milk production. Frequently the change in environment is sufficient to reduce it temporarily.

Cost of Milk. The wet-nurses in Sarah Morris Hospital receive their board and room and $12.00 per week. Figuring the former at $10.00 per week, this would total a cost to the institution of $22.00 per week for each nurse. With an average of 30 to 40 ounces of milk per nurse daily, or 210 to 300 ounces per week, the average cost will be about 7 to 10 cents per ounce, or approximately $2.25 to $3.25 per quart.

When milk is dispensed to patients outside of the hospital, a charge of 10 cents an ounce is made for it, which is a reasonable price when all of the contending factors are taken into consideration.

Number of Nurses Needed. Each good wet-nurse can care for the needs of about two infants, depending upon their weight and development. In addition her own infant can often be satisfied with the strippings. When insufficient her baby may be given a mixed diet.

Length of Lactation. No time-limit is placed upon the employment of a wet-nurse as long as the quality and quantity of her milk is sustained, and she continues in good health. One of our nurses had an infant eighteen months old. Such long periods of lactation, however, as a whole are not to be advised.

The Wet-nurse's Baby. The presence of the wet-nurse's baby predisposes to her peace of mind, and wherever possible, she should take it with her. Her baby's state of health is by all means the best indication as to her ability as a nurse, and, with this, the presence of constitutional disease in herself. It may be of immense value, if the baby is strong and healthy, to keep up the flow of milk, in case the baby to be nursed is a weakling. It may also be used to estimate the functional capacity of a wet-nurse by nursing at regular intervals, and weighing before and after the nursing for twenty-four-hour periods. If in perfect health, it may be put to the breast, after the weakling has taken such milk as it has strength to draw. If this is not practicable, then the weakling should be nursed alternately with the well baby on each breast. It is also of immense value in emptying the breast after the wet-nurse has removed as much milk as it is possible by expression or by the breast-pump, if this is the means of drawing the milk for the weakling. It is a well-known fact in all institutions where wet-nurses are used, that the greater the degree to which the breasts are stimulated by suckling infants, the greater will be the reward in production. If the milk is insufficient for both babies, partial or entire meals of artificial food may be substituted for the wet-nurse's infant.

At the first sign of an acute illness on the part of the wet-nurse's baby, it should be separated entirely from the other baby, and removed from the breast; its illness should be given the same serious consideration as that of the other infant, so that the mother's anxiety may be relieved. It should receive as much of its mother's milk as can be spared. This can be expressed from the breasts and fed from a bottle.

Feeding of the Wet-nurse's Baby. When a single infant is to be nursed, the second baby is often a necessity in the promotion of the development and stimulation of her breasts. No breast can be developed to its fullest capacity with the breast-pump or hand expressions. It is a well-known fact that the breasts will respond in proportion to the demand placed upon them, and in most instances during the first few weeks of the premature's life, when its demands are met by from 4 to 16 ounces of milk, the wet-nurse can supply sufficient milk for both babies. When her supply becomes insufficient to meet the demands, her baby can be put upon partial bottle feedings of the strength as indicated by its age and development. The progress of the wet-nurse's baby has great influence on her peace of mind, which may spell success or failure in her ability to carry out her work. When the premature infant gives evidence of sufficient strength to be placed upon the breast, we have found the application of the wet-nurse's baby to the other breast a very valuable expedient in aiding the flow of milk into the breast which is to be nursed by the weakling. In many instances we have seen the milk flow from the second breast by this method so freely that but very little effort was required on the part of the weakling to obtain its food.

2. The Hygiene of the Wet-nurse.

In general, everything that has been said in the chapter on hygiene of the nursing mother applies also to the

wet-nurse—of course, with the proper modifications, made necessary by peculiarities of her position.

Her clothes should be simple, and in every part washable. As the care of her undergarments is of even greater importance than her outer clothing, it is well that her laundry should be done with the family work, so that the family laundress who is trusted by the family may be charged with its inspection.

To simplify nursing or the drawing of milk, the author has devised two garments for wet-nurses. The material used for the outer garment is of yellow gingham, such as is used in the making of hospital uniforms, the yellow color being selected to distinguish the wet-nurse from the blue, as used by the nursing corps. The corset-waist is to be made of heavy muslin. The corset, if worn at all, should be of a very low type, so as to avoid all pressure on the breasts. It is best of a cheap quality, so that it can be replaced frequently for sanitary reasons. Each wet-nurse should be supplied with four uniforms and six nursing corset-waists.

The Diet of the Wet-nurse. There is danger of the creation of indolent habits through neglect of regular exercise and the lack of regular household duties, but even greater danger lies in the direction of overfeeding with unusual foods. The average wet-nurse is either obtained from an institution or a home in which the luxuries of life are limited, and she has been accustomed to a simple nutritious diet. Every attempt should be made to supply the nursing woman with a well-rounded diet of simple foods, with milk and cereals as the basis, and these supplemented with meats, soups, the common vegetables, limited amounts of fruits and plain desserts. In so far as possible, the aromatic vegetables, unripe and highly acid fruits, fried meats, and rich pastries are to be avoided. We believe that, on the whole, too great stress has been laid upon the danger of the diet in the mother of a full-term infant, and in most cases the average

mother can partake of a very full diet. However, in the case of the woman nursing premature infants, it should become a custom to allow only such foods during the first few days after her installation as can be given with perfect impunity. When a full, free flow of milk is established, other vegetables and fruits can be added, one at a time, and after each addition to the diet a try-out should be given the milk. We have on numerous occasions seen marked intestinal distention and diarrheal attacks following even seemingly slight indiscretions of the diet on the part of the wet-nurse. It is our hospital practice to furnish each wet-nurse with two quarts of good wholesome milk daily, and at least one pint of cereal gruel, preferably farina or corn-meal. A mixture of milk and cereal gruels makes a very good combination for drinking midway between meals. The remainder of the milk may be taken with the meals, either pure or in the form of cocoa, tea, or weak coffee, in whichever form it is best taken by the individual woman. The latter is of considerable importance, as in the forced diets which are required, where an abundance of milk is demanded, distasteful foods soon become obnoxious in large quantities.

Beers, malt-extracts, and other rich drinks are not forced upon the nurse, unless she is accustomed to them, and feels their need. It must always be remembered that an excess of fluids would naturally tend to dilute the milk unless the secreting gland be of exceptional development.

Exercise of the Wet-nurse and Her Work. She should be impressed before her engagement with the fact that she will be required to do a moderate amount of work and exercise regularly out of doors. The former will be of service in promoting her general health, and both the work and the exercise will serve as a nerve tonic and prevent her becoming indolent. This does not mean that she should become a drudge, but that she should at

least be required to care for her own room and her own infant's clothes, and should be made to feel that in return for her laundry work she would be requested to do some light general work about the house. Her exercise in the open air should so far as possible be at regular times. The question as to the care of the napkins of both babies is open to considerable discussion; and it may be stated that whenever it becomes necessary for the nurse to express her milk by hand, she should not be subjected to the handling of soiled napkins, whenever this can be averted.

Other Conditions Influencing the Quality of the Breast Milk.

The nervous and mental state of the nurse is of the utmost importance, and wherever possible an emotional, nervous, erratic woman should be excluded, because of the tendency of these influences to suppress the flow of milk. Therefore, whenever possible, a woman of more or less phlegmatic temperament is to be selected. This is especially true in the case of a woman who is to be in close contact with and is to nurse an infant with neurotic tendencies. There is also the possibility of the same influence being manifest in time of slight indisposition on the part of her own infant, and such an individual is also more likely to resent the necessity of partial or entire artificial feeding of her own child to the advantage of the premature infant, when it has reached such an age when it may make greater demands on her supply.

Menstruation rarely produces any serious disturbances. It is always a safe procedure to dilute the milk during the first and the second day of menstruation when the nurse suffers considerable pain at these times.

Period of lactation may or may not be a considerable factor, depending upon the individual woman. We had in our employ a nurse who had been with the institution for sixteen and a half months, and whose infant was

eighteen months old, and who supplied us with the largest quantity and the best quality of milk of the four nurses in the institution.[1] When possible a nurse should be selected after the first few weeks of lactation, at which time the colostrum has disappeared from the milk, and the quantity and quality of her milk has become established. After the first few weeks of lactation, but little or no attention is to be paid to the age of the wet-nurse's baby as compared with that of the infant to be fed, and we have never noted any ill effects following this rule.

3. The Nursing.

The Infant's Bedroom. Under ideal circumstances, this should be separated from that of the wet-nurse. This is especially true where a trained attendant has care of the infant. It should under all circumstances also be separated from the wet-nurse when she is of a low degree of intelligence and of a type not to be trusted with the care of the infant.

Methods of Drawing Milk. Numerous methods of obtaining milk from the breasts have been described, but only those most practicable of application will be detailed. These should be divided, first, into those in which the baby is placed directly at the breast, and those methods by which the milk is drawn from the breasts and fed to the infant. Two methods are especially applicable

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[1] The milk of this nurse was examined in the laboratories of the University of Chicago after seventeen months of lactation with the following result:

Albumin	1.30	per cent.
Casein	0.69	" "
Fat	3.54	" "
Lactose	7.025	" "
Salts	0.1885	" "

It must be remembered that this is an exceptional case, and but few women under the stress of ordinary life can properly nurse their infants after the ninth to twelfth month.

where the baby is fed directly on the breast, and needs assistance because of its weakness.

1. Premature infant is placed at the breast, and is supported there by the nurse's right arm while nursing at the right breast, and the left hand is used to grasp the breast just above the nipple between two fingers (see

Fig. 1.—Proper method of holding baby during nursing.

Fig. 1), and the milk is expressed directly into the baby's mouth. In this way the baby is taught to take the breast, and at the same time receives its food with little effort. This method can be continued until the baby has gained sufficient strength to nurse without assistance.

2. Much of the same result can be accomplished by placing the wet-nurse's baby on the opposite breast during the nursing period, whereupon the simultaneous nursing on both breasts will cause a free flow of milk into both sides.

In those methods by which the milk is drawn from the breasts and fed to the infant by hand or by other means.

1. By the breast-pump. The modification of Holz vacuum apparatus, as devised by the author, by which means the milk is drawn directly into two graduated 2-ounce flasks, which can be filled to the quantity desired, and stoppered for future use, so that the milk is free from handling, and thereby avoid contamination.

2. By direct expression, which is performed as follows:

Fig. 2.—Author's improved breast milk collector. The pump is made in two types, the first filled with a large rubber bulb of a size considerably larger than is ordinarily sold with a breast-pump, and the second with an attachment to which the Holz vacuum pump can be fitted. In place of the ordinary collecting bulb at the lower surface, an arm is so constructed as to allow the milk to drain into specially designed graduated 2-ounce milk flasks.

Instructions for the Expression of Breast Milk.

Scrub the hands and nails with soap, warm water and a nail brush for at least one full minute. Wash the nipple with fresh absorbent cotton and boiled water or a freshly made boric solution. Dry the hands thoroughly on a clean towel and keep them dry. Have a sterilized

graduate glass tumbler or large mouth bottle to receive the milk.

1. Grasp the breast gently but firmly between the thumb placed in front and the remainder of the fingers on the under surface of the breast. The thumb in front and the first finger beneath should rest just outside of the pigmented area of the breast.

Fig. 3.—Direct expression of milk (act 1).

2. With the thumb a downward pressing motion is made on the front against the fingers on the back of the breast, and the thumb in front and fingers behind are carried downward to the base of the nipple.

3. This second act should end with a slight forward pull with gentle pressure at the back of the nipple which causes the milk to flow out.

The combination of these three movements may be described as "Back-down-out."

It is not necessary to touch the nipple.

This act can be repeated thirty or sixty times per minute after some practice.

Fig. 4.—Direct expression of milk (act 2).

Both breasts may be emptied if necessary or they may be used alternately.

The milk should be covered at once by a sterile cloth held in place by a rubber band and kept on ice until used.

Daily Number of Expressions. Expression is performed six times daily at regular intervals of four hours during the day and night.

CHAPTER IV.

THE NURSING INFANT.

Signs of Successful Nursing. The normal full-term infant shows a gain of not less than 4 ounces weekly. This is the minimum weekly gain which may safely be allowed. When a nursing baby remains stationary in weight or makes a gain of but 2 or 3 ounces a week, it means that something is wrong, and the defect will usually, but not invariably, be found in the milk supply. When the baby is nursed at proper intervals, and the supply of milk is ample and of good quality, it is satisfied at the completion of the nursing. Under three months of age it falls asleep after ten or twenty minutes at the breast. When nursing period again approaches, it becomes restless and unhappy, crying lustily if the nursing be delayed. When the breast is offered, it takes it greedily. The weekly gain in weight under such conditions is usually from 4 to 8 ounces. At the fifth month the baby will have doubled, and at the twelfth month trebled its birth weight. The average gain per week during the first year is about 4 ounces.

The baby increases in length from about 20.5 inches (50 cm.) to 28.5 inches (70 cm.) in the first year. The first tooth appears at about the sixth or seventh month, and at one year there should be six teeth or more. (Age in months minus 6 = number of teeth normally present at that age.) It begins to smile at about the fifth week, grasps objects and holds its head erect in the fourth month, sits alone for a few minutes at seven or eight months, bears its weight on its feet at the ninth or tenth month, stands with slight assistance at the eleventh or twelfth month, and creeps or walks soon after this (tenth to eighteenth month, average fourteenth month), and says a few words towards the end of the first year.

Stools. *The feces* of breast-fed babies are *strikingly uniform,* and are *like no other bowel movement in in-*

fancy. Normally, there are two or three a day, some times only one, or even more than three. They are soft, or mushy, homogeneous, of an egg-yellow or gold color, and have a slightly sour, not at all unpleasant odor. They are never formed, and always cling to the diaper. The nature of the bowel movement, and its uniformity, is due to the "physiological fecal flora" which is brought about by the ingestion of breast milk into the germ-laden intestinal tract, and which in turn have a fermentative rather than a putrefactive action on the food. The gases normally formed are carbon dioxide and hydrogen, and these are practically odorless. The acidity of the movement, its softness, and the mechanical action of the gases present, all insure active peristalsis and ready emptying of the bowels, so that constipation is an exceptional condition in a breast-fed baby, and, if present, it nearly always suggests too little food, or abdominal and intestinal muscles too little developed and too weak to force the stool past the anal sphincter. This latter condition is commonly interpreted as constipation by the laity.

The dried residue of the feces contains from 10 to 30 per cent. of fat, about 8 per cent. salts, a very large percentage of bacteria, bile pigments, intestinal secretion (mucus, etc.), epithelial cells, etc. No food proteins or carbohydrates are found.

The feces of the breast-fed baby are very frequently not wholly normal; they quite commonly, especially during the first few months, contain small, soft, white or yellowish fat curds, an excess of mucus, and are often greenish in color, and may be more frequent than normal. *Such a condition is perfectly consistent with a normal growth and well-being of the baby, and should never in itself be a cause of worry, or an indication for a change of food.* This is a very important point that is very commonly neglected. The condition of the bowel movements is only one factor, and in the breast fed a minor one, in determining a baby's nutrition.

CHAPTER V.

MIXED FEEDING AND WEANING.

Mixed Feeding (allaitement mixte). With a diminution in the amount of milk secreted, the breast milk must, of course, be complemented or supplemented by modified cow's milk. These methods of feeding are usually successful. By *complemental* feeding we mean the administration of milk from a bottle following a period at the breast at each nursing. By *supplemental* feeding substitution of a bottle for a breast feeding is meant. Thus, in the former the baby receives as many part bottle as breast feedings, while in the latter it will be supplied with one or more bottle feedings to replace breast feedings. As we know that the breast secretes in proportion to its stimulation, the complementary feeding is far more satisfactory, and not infrequently it is wise to nurse both breasts for a short time, let us say, each one five to ten minutes, before the bottle is given.

It is necessary to weigh the infant before and after nursings for one or more days to obtain a fair idea of the amount of cow's milk mixture that it will be necessary to administer in cases of underfeeding at the breast. Given a normal infant weighing ten pounds, the food value of twenty-five ounces of breast milk should be approximated ($\frac{1}{6}$ of his body weight).

Clinical experience has taught us that most infants will thrive on artificial mixtures approximating the food values contained in breast milk. Roughly, one and one-half ounces (45 mils) of cow's milk to which $\frac{1}{10}$ ounce (3 grams) of sugar, cane or milk, and one ounce of water has been added, will meet the requirements for each pound body weight ($\frac{1}{2}$ kilo). Such a mixture will contain F-2.4, P-2.1, C-6.7 per cent.

(63)

Example: A ten-pound infant receiving 15 ounces of breast milk is receiving two and one-half ounces for each six pounds of his body weight and will require in addition the equivalent of two and one-half ounces for each of four pounds body weight which can be supplied by six ounces (one and one-half times four) of cow's milk and four-tenths of an ounce of sugar.[1] To meet his water requirements, the mixture should be made 10 ounces, adding four ounces of boiled or cereal water.

The cow's-milk mixture is best fed in part after each breast nursing. By giving both breasts at each feeding they receive the maximun amount of stimulating. The infant may be fed at three- or four-hour nursing periods.

For the thin infant breast milk plus substitute feedings must approximate the requirements of his full weight for his age.

In beginning the use of cow's milk, however, it must be remembered that at first a weaker strength must be used than the child will require for growth, this weaker food being necessary in order gradually to accustom the infant to the change. If too strong a cow's milk mixture is given at first, it will be very apt to disagree, causing colic and vomiting. Later, when the child has become accustomed to the new food, a stronger mixture may be given. When a mother cannot give her infant at least two satisfactory breast feedings daily, it is advisable to wean the child. The newborn baby is not very discriminating, and will nurse anything equally well. The older baby, however, quickly prefers the easy-flowing bottle to the increasingly unsatisfactory breast, and will quite regularly stop nursing at the breast as the milk comes harder and is less abundant. If the bottle is given right after the breast, it is always well to use a nipple from which the milk comes with some difficulty, for the reasons given above. If it

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[1] Cane sugar—two level tablespoonfuls equals one ounce. Milk sugar—three level tablespoonfuls equals one ounce.

is desirable to wean the baby rather quickly, this method of following the breast by the bottle is often to be preferred to the other.

Indications for Weaning. *Pregnancy* is usually an indication for weaning. The mother's milk becomes more scanty, and often poor in quality. This is especially the case if the mother knows she is pregnant, and has been taught that a pregnant woman should never nurse a baby. If the baby continues to thrive at the breast, there is no reason why nursing should not be prolonged. Fortunately a new pregnancy does not often supervene before a time that makes it quite safe to wean the nursing baby, *i.e.,* before the sixth month.

In acute infections in the mother, such as *pneumonia,* and the acute contagious diseases, such as *scarlet fever,* one must weigh the danger from exposure to infection as against the quality of the artificial food and environment in the individual case.

In the milder contagious diseases, such as *measles, mumps,* it is true that young breast-fed infants are rarely infected. *Pertussis* is an exception, and has a high mortality in the newborn and young infants; and the infant should under all circumstances be protected from exposure. In the presence of *diphtheria* the infant can be immunized with safety.

Weaning should always be done gradually, when possible, for the sake of both mother and the child. In cases of sudden weaning, the food must be very much weaker in the beginning than for an artificially fed child of the same age. If weaned at six months, the infant should be put on a mixture suitable for a child of two or three months, and the same rule applies for older infants. When the infant becomes accustomed to cow's milk, the strength can gradually be increased. Rarely should breast feeding be continued beyond the first year.

The fear of the laity of the "second summer" is well founded when dirty milk and other improper foods are

fed promiscuously, but with clean, certified, and sterilized milk, and properly prepared soft foods, the dangers of the summer heat are minimized. It should be our rule to underfeed rather than overfeed in hot weather, and during the hot spells the infant's diet may well be reduced one-half.

Care of the Breasts During Weaning. When the breast feeding is carried on the usual length of time (from nine to twelve months), the process of weaning ordinarily causes little or no discomfort. All that is usually required is to press out enough of the milk to relieve the patient as often as the breast becomes painful, which may not be more than two or three times a day. When the weaning is necessarily abrupt, no little discomfort may result. When the weaning can be accomplished more gradually, the infant should have one less nursing every second or third day, until only two are given daily. After this has been practised for one week, nursing should be discontinued. In cases of sudden weaning, a saline laxative, such as citrate of magnesia or Rochelle salts, should be given every day for five days—sufficient to produce two or three watery evacuations daily. In the meantime the mother should abstain from fluids of all kinds up to the point of positive discomfort. The breasts should be elevated by a firm binder.

CHAPTER VI.

NUTRITIONAL DISTURBANCES IN THE BREAST-FED INFANT.

BREAST milk alone furnishes all of the needs for growth and development of the human offspring. The infant will thrive in most instances on breast milk from different sources and different quality, demonstrating the ability of the average infant to assimilate the food which Nature intended for its use, even though the percentage quantity of the various components may vary greatly. Disturbances in the breast-fed baby are dependent upon one or more of several factors. In the order of their frequency they may be divided, as follows.

1. Underfeeding.
2. Overfeeding.
3. Congenital debility, with resulting impairment of the vital functions.
4. Intercurrent parenteral (pharyngitis, tonsillitis, bronchitis, pneumonia, pyelitis, etc.) and enteral infections.
5. Idiosyncrasy towards mother's milk.

While all nutritional disturbances in young infants are of serious import, they are far less dangerous than those of the artificially fed infant, and much more easily corrected. They are also much less frequent than nutritional disturbances in artificially fed infants.

1. Underfeeding.

Etiology. Two factors of prime importance must be investigated to complete the diagnosis:

(1) The daily quantity of the milk furnished to the infant.

(2) The quality of the milk supplied by the mother.

(67)

The milk may contain the normal percentage of fat, sugar, and protein, but be scanty in amount. Instead of the 4 or 5 ounces to which the child is entitled, it may get but 1 or 2 ounces. Whether or not the quantity is sufficient, may be determined by weighing the baby before and after each nursing for twenty-four hours. (The ordinary spring balance infant scale will not answer, and a simple beam scale with weights and scoop should be supplied.) One ounce of breast milk weighs practically 1 ounce avoirdupois. By nursing for fifteen minutes, a child under one week old should gain 1 to 1.5 ounces; at three weeks of age, 1.5 to 2 ounces; four to eight weeks of age, 2 to 3 ounces; eight to sixteen weeks of age, 3 to 4 ounces; sixteen to twenty-four weeks of age, 5 to 7 ounces; six to nine months of age, 6 to 8 ounces; nine to twelve months of age, 8 to 9 ounces. Of course, arbitrary limits cannot be fixed as to the quantity. It is not necessary to worry about the quantity taken at individual feedings so long as the infant is making satisfactory gains in weight, and the general progress is good.

Quantity of Human Milk Required by the Nursing Baby. Babies of the same age and weight, under the same conditions, will take nearly the same amount of food. The older and larger the baby, the larger the total quantity of food required, but its *energy quotient*—that is, the number of calories per kilogram or a pound of weight—lessens steadily with increasing age. The daily amount that normal, thriving babies take from the breast can be stated at about one-sixth to one-fifth of their body weight during the first month, about one-sixth to one-seventh up to the sixth month, and about one-eighth after the sixth month. Heubner expressed this in terms of energy quotient, as follows: "During the first few months an infant requires 100 calories per kilogram daily of breast milk; after the sixth month this energy quotient gradually comes down to 80 or 85 at the end of the first year. An

energy quotient of 70 is about the minimum amount that an infant can take without losing weight." Human milk can be estimated at 21 calories per ounce, and about 70 calories per 100 Gm. of milk. With these figures in mind, it is easy to determine whether a breast-fed infant gets about the right amount of food, and we have also a valuable standard by which to measure the food of an artificially fed infant.

Symptoms. Failure to gain weight properly, or even a loss in weight, may be the first positive evidence of an insufficient food supply. Usually this is associated with more or less evidence of dissatisfaction on the part of the infant. The infant's sleep becomes disturbed, and it becomes restless, and cries long before the next feeding time. Again, it may manifest its dissatisfaction by nursing greedily for a short time, releasing the breast and crying. It returns to the breast again, but with the same result; or in other instances the infant will remain at the breast for much longer periods than should be necessary to obtain the food that it needs, which should be accomplished in from ten to twenty minutes.

Usually the stools are normal in appearance, but small in amount, and give little evidence of the cause of the trouble. However, if the food supply be decidedly insufficient, we may have a positive evidence of the underfeeding by the appearance of the so-called "hunger stools," which are of more or less brownish or greenish-brown color, containing little fecal matter and much mucus.

If the condition is not corrected, the baby becomes weak and apathetic, the skin loses its turgor, the temperature becomes subnormal, it is pale and anemic, the fontanelles become depressed, and the abdomen sunken. Whenever there is room for doubt as to the cause of this group of symptoms, the scale will be the most positive evidence.

Treatment. Undue haste in removing the baby from the breast offers the greatest danger in the treatment of underfeeding, and should be resorted to only when other means fail.

Mixed Feeding should be instituted when the breast milk supply is inadequate. (See page 63.)

The ability to increase the quantity of milk secreted by the average woman must necessarily vary directly with the quantity and quality of the glandular tissue composing the breast. However, to a certain extent at least, certain factors will more or less directly influence the quantity and quality of the secretion, and they are worthy of our attention.

Means of Stimulating the Breasts. The *surroundings* of the mother must predispose to a happy frame of mind; she must not be overburdened with household cares; her exercise must be regular, and she must be relieved of worry and lack of sleep. It is well, if possible, to free her from all care of the baby, especially at night. She should be put in as good physical condition as possible; she should get out of doors.

Giving both breasts at each feeding and temporarily shortening the nursing periods to 3 or even 2 hours, thereby increasing the number of feedings, are the best stimulants available.

Her *appetite* should be stimulated, so that she will take an abundance of milk and other nutritious food. The very common forced feeding beyond the natural appetite, is of questionable value. The general rules as to the diet previously spoken of should be maintained. It should, however, be remembered that an excessive diet may be assimilated by the mother's body without increasing the flow of milk. The fluids given should be palatable to the nursing mother, and, as previously recommended, milk, weak tea, cocoa, farina, oatmeal, and cornmeal gruels as well as milk soups are probably the best. The fat and the protein of the milk can more especially

be influenced by the diet. The fats are increased by over-feeding with fats and carbohydrates, with little or no exercise. They are reduced by limiting these articles and substituting vegetables, and by increasing the amount of exercise. The protein is also increased by overfeeding and limited exercise. The carbohydrates are less influenced by the diet, but are also affected by an excess of carbohydrate feeding. Alcohol in the form of malted drinks has a temporary influence in increasing the quantity of milk and the amount of fat. The effect on the protein is less constant. We never force a woman to partake of alcoholic liquors unless she desires them, because of the moral as well as of the physical effect.

Direct Expression is the best means of breast stimulation.

Stimulating *massage* may be applied to the breast in such a manner as to stimulate the whole gland. This can best be accomplished by two movements: (1) by gently raising the whole breast from the chest wall and kneading it gently between the fingers, and (2) by holding the breast against one hand and making circular movements around the periphery with the outspread finger tips of the other hand, and gradually working from its base towards the nipple.

Baths at a temperature comfortably cool (80° to 90° F.) should be taken daily to promote her general health as well as cleanliness. These should be followed by a brisk rubbing with a coarse towel.

Steaming the breasts by the application of hot towels covered with oiled silk two or three times daily is of decided benefit.

Galactagogues of any material value for permanent use are unknown. Pituitrin has been recommended for temporary stimulation. We have not had much experience in its use. General tonics will often improve the digestion and tend to overcome the anemia, and in this

way improve the general health, and thereby lactation. Feeding dried placenta has been recommended.

2. Overfeeding.

This condition is a rare one in the breast-fed baby, and, when present, in all but the very young and premature, nature often provides its own remedy, either by regurgitation on the part of the baby, or by its refusal to nurse longer than to meet its needs, which latter soon leads to a lessened milk secretion. In the first weeks and months it may be of considerable importance, and may cause grave symptoms on the part of the infant—that is, before the mother's breast and the infant have become adapted to one another.

Etiology. Although overfeeding in the breast-fed infant is rare when compared with overfeeding on artificial food, yet next to underfeeding it is the most common form of nutritional disturbance in the breast-fed infant. It is also more commonly present in infants fed by a wet-nurse than in infants nursing the maternal breast.

Usually the error lies in too frequent nursing.

Rarely it may be due to excessive quantities of milk taken at proper intervals.

Occasionally it is due to milk which is excessively rich in fat.

Pathogenesis. The normal infant's stomach on breast feeding empties itself in about two hours. When all the food has left the stomach, and is undergoing intestinal digestion, free hydrochloric acid is forming in the stomach. Free hydrochloric acid is antiseptic, and it also stimulates secretion of pancreatic juice and secretion of bile, both of the latter products being essential to proper intestinal digestion.

For normal digestion it is therefore necessary that the stomach remain empty for some time after all the food has left it. When by too frequent nursings no time is

allowed for the above described physiological process, or when by excessive quantities of food at proper intervals too great demands are made upon the hydrochloric acid, and the time of gastric digestion lengthened, with corresponding shortening of the period of comparative rest, or the gastric secretion diminished by excessive fat, then we may expect disturbance of the normal digestion due to overfeeding.

Symptoms. The earliest symptoms are regurgitation, diarrhea, and lessened appetite. These three symptoms are reactions of the organism to excessive intake of food attempting to get rid of the excess.

Regurgitation occurs at first occasionally only, immediately after nursing, and without any discomfort on the part of the infant ("spitting"). The regurgitated fluid is often unchanged milk. This is usually the first premonitory symptom.

Diarrhea follows when overfeeding continues and regurgitation becomes insufficient to rid the body of excess of food. The stools are more frequent than normal, and contain undigested particles of food.

Lessened appetite, although present in many cases, may be replaced by symptoms suggestive of hunger, the infant taking the breast and nursing greedily. This apparent symptom of underfeeding and of hunger may wrongly be interpreted, and lead to additional overfeeding by giving the breast at even more frequent intervals to allay the apparent hunger and to quiet the restless infant.

In many cases no other symptoms develop, the condition undergoing a spontaneous cure. The breasts lessen their yield, and thus the cause of the condition disappears, or, on the other hand, the digestive power of the infant increases to such an extent as to be able to take care of the excess, if not too large. This accounts for the fact that frequently the above-named symptoms are neglected, since they usually produce improvement in

the child's condition, and are regarded as passing disturbances without much importance. When, however, they are entirely neglected, and excess of the food is continued, or even increased, due to wrong interpretation of symptoms, then more serious symptoms develop, and the condition reaches a stage where spontaneous cure rarely occurs.

Vomiting becomes habitual, occurring from a few minutes to half an hour after nursing. It is accompanied by visible discomfort and straining on the part of the infant. The vomitus consists of curdled milk, mucus, and gastric juice. Between vomiting there is often painful belching. Stomach shows distention, and empties itself only after three to four hours. Free hydrochloric acid is almost or entirely absent, the acid products of fermentation being present. The micro-organisms are increased in number and variety, due to stagnation and absence of antiseptic free hydrochloric acid.

Initial diarrhea is sometimes followed by temporary constipation, diarrhea setting in again. The evacuation is painful, and, with much gurgling and discharge of gases, fluid masses are squirted from the anus. The stools are watery, with white and dark green fragments, and of disagreeable, sour, pungent odor. The irritating feces often causes eczema and intertrigo in the anogenital region.

Abdomen is distended, tense, and often there is visible peristalsis. Intestinal colic causes restlessness and crying; the infant's face gives expression to its pain, and, as the fermentation increases, its agony is increased, due to intestinal paresis.

The infant becomes restless; its sleep is much disturbed, and even during sleep its features give evidence of its distress.

The weight early becomes stationary, and in severer cases associated with dyspepsia loss of weight becomes marked.

Complications. *Dyspepsia.* Accompanied by the milder evidence of intestinal irritation, evidenced by increased peristalsis, with its resultant colic, more or less numerous bowel movements of eight or ten or even more daily, sour and irritating, greenish-yellow in color, and containing numerous curds and much mucus. The buttocks soon become reddened and intertrigo results.

Intoxication, while rare in the breast-fed infant, may result when the dyspepsia is neglected. The baby becomes drowsy and stuporous, paying little attention to its surroundings, and not infrequently develops a severe anorexia, all associated with more profound intestinal symptoms.

In dyspepsia the intestinal findings dominate the picture, while in intoxication they share their prominence with the added nervous symptoms.

Pyelitis is not an infrequent complication in neglected dyspepsia and intoxication, and while it undoubtedly is frequently due to an ascending infection, it may result from extension through the blood stream or the lymphatics.

Eczema not infrequently results from overfeeding in the breast-fed infant, and is usually seen in the fat type of infant who is otherwise healthy.

Pylorospasm and gastric dilatation are not uncommon in the neglected cases.

Acidosis may develop in the extreme cases, associated with great loss of weight, but this is rare.

Diagnosis. In the presence of symptoms suggestive of overfeeding, positive diagnosis is made by determining exactly the amount of milk taken by the infant, and comparing this amount with what an infant of the same weight and of the same age should get. The method of this determination has been described in detail under the treatment of underfeeding.

If, however, the food is found to be quantitatively correct, occasionally information of value may be obtained

by examining the quality of the milk chemically, especially as to its fat content. The specimen for examination should be taken under precautions pointed out under Examination of Human Milk. By making proper etiological diagnosis, valuable indications for rational treatment are obtained.

If a careful search is made for the etiological factors in the common illnesses of infants, which are so frequently charged to overfeeding, one will be surprised to find that the error lies in the diagnosis, and that in most cases the condition is not due to overfeeding. This leads us to warn against the only too frequent habit of weaning infants without a careful study of the exact cause of the infant's trouble.

Treatment. *Prophylaxis* of this disturbance is of importance, and consists of giving the nursing mother proper instructions as to the nursing, especially as to its frequency, and seeing to it that the rules for nursing, as laid down elsewhere, are observed by the nursing mother In wet-nursing, more caution is necessary, especially in those wet-nurses who have an abundance of milk, which is frequently the case in a wet-nurse whose own child is much older than the infant to be nursed.

A very important point to impress both on the mother and also on the wet-nurse is the fact that crying of the infant is not always due to hunger, and that offering the breast should not be used as a means for quieting the child.

When the initial or mild symptoms only are present, then correction of the nursing habits is usually sufficient, the infant improving without any special treatment.

When the error lies in too frequent nursings, it is best and often completely relieved by lengthening the feeding intervals to three or, even better, four hours.

It is of equal importance that the infant should not be left too long at the breast. The best average nursing time being about fifteen minutes, with twenty minutes as the

maximum. However, when the flow of milk is very free, it may be necessary to reduce the nursing period to even three to five minutes, it being a fact that most infants take about 75 per cent. of their entire meals in the first five minutes at the breast. It is always well at the beginning of such an experiment to weigh the baby after a two, three, five, ten, and twenty minutes period to ascertain the exact amount which the baby obtains from the particular breast which it is nursing, so that conclusions may be drawn definitely as to the time it is to be left on each breast.

If placing the infant at the breast for short periods with long intervals does not give results, it is advisable to express the milk, and feed in small quantities from the bottle. And if another baby be at hand, it may be placed upon the breast to keep up the supply. Or when a wet-nurse is available for temporary use, the babies may be exchanged.

Weaning should under all circumstances be considered · only as the last resort, after all other methods of adapting the infant to the breast have failed.

An excessive amount of fat in the milk is more often due to an excessive intake of food in general on the mother's part than an excess in any one element, and can be diminished best by cutting down the food as a whole, lessening the amount of all food.

When the condition has progressed farther, and the symptoms have become more serious, then it is necessary to treat the infant also. The treatment consists in emptying the stomach and the bowels of the overload of fermenting food, and of rest for the digestive apparatus, both these objects being achieved by giving a bland diet, consisting of boiled water or weak tea sweetened with saccharin, for twelve hours, the digestive tract getting rid of its contents spontaneously.

If the symptoms improve upon this treatment, the nursing should be gradually resumed by giving two

breast feedings in the twenty-four hours following the period of starvation, substituting for the other nursings bland liquids, and increasing cautiously the number of nursings.

If on withholding the food, vomiting does not cease, then it is necessary to wash out the stomach.

Irrigation of the bowel is often necessary, and aids in removal of fermenting intestinal contents, and allows also the gases to pass, thus relieving the distention and colic. Only when change of diet and irrigation are not sufficient, then the use of purgatives is advisable, castor oil being just as efficient and less harmful than the frequently preferred calomel.

Colic usually disappears on correction of the diet, and after the intestinal tract has been cleansed of its irritating contents, and of gas. Massage to the abdomen will aid the passage of gases which cause distention, when the bowels tend to become paretic. In severe pain, warm applications to the abdomen give relief. If these measures fail to bring relief, and the pain is such that the infant is deprived of sleep, a mild sedative in small doses may be given.

Feeding of powdered casein in amounts varying from 6 to 8 Gm., dissolved in 30 to 60 mils of water, two or three times daily will relieve colic in many infants, in all probability due to lessening of intestinal peristalsis.

There is a class of infants who, although they are gaining progressively in weight, cry a great deal, expel a great deal of gas, and perhaps have a green stool now and then. It is almost criminal to take such infants off the breast, although the temptation to do so is very great, because of the worry they cause the mother, and consequent harassing of the physician. Such an infant will frequently cry for six, eight, ten, or twelve hours out of the twenty-four, and still make a good gain in weight each week, in which case it is very probable that the infant is being overfed, and the food supply should be

reduced. The mother's diet and general habits should receive attention.

3. Congenital Debility, with Resulting Impairment of Vital Functions.

Etiology. Premature birth is the most important condition causing debility associated with deficient functionating power of the digestive organs. Method of feeding premature infants will be detailed later in a special chapter.

Hereditary weakness of the offspring caused by disease in the parents is frequently the cause of deficient morphological and functional development of the digestive organs, and thus it is often the underlying cause of nutritional disturbances, which are more commonly chronic in character. Tuberculosis, syphilis, and alcoholism in parents stand at the head of the conditions causing hereditary weakness, even when the offspring does not inherit the disease itself.

Malformations of the digestive tract (cleft palate, sublingual tumors, pyloric stenosis, atresias of the intestinal tract, Hirschprung's disease, etc.) from any cause compromise its functional capacity usually, but in most cases they cause serious conditions necessitating surgical interventions, and only rarely do they produce simple nutritional disturbances amenable to dietetic means, and therefore they belong to the domain of surgery.

Symptoms. As may be expected, symptoms of these so diverse conditions vary. Hereditary weakness may often be suspected when symptoms of nutritional disturbances develop even when the infant is given the best care possible, and the milk is quantitatively and qualitatively correct. Symptoms of underfeeding or of overfeeding, as described previously, may be present, depending upon the etiological factor.

Diagnosis. Careful examination for malformations, and thorough family history, in cases of suspected

hereditary weakness are of chief importance in making the etiological diagnosis.

Treatment is usually determined by the pathology. and by the nature of the particular nutritional disturbance which developed.

4. Intercurrent Parenteral and Enteral Infections.

Etiology. Diseases both in the mother and in the infant are to be considered in etiology of this condition. In the mother the most important are the general infectious diseases, *e.g.*, puerperal fever and sepsis, typhoid, pneumonia, etc., and local infections of the breast, and also of the upper respiratory passages. In the infant there are parenteral infections, that is, infections outside the digestive tract, *e.g.*, pharyngitis, tonsillitis, pneumonia, pyelitis, bronchitis, and enteral infections, or infections of the intestinal tract, which will be discussed under a special heading.

Symptoms. In the conditions dependent on the mother's health the symptoms will vary first with the quality and quantity of her milk supply, which will have an effect on the child's general nutrition, and, secondly, may result in direct parenteral or enteral infections of the infant.

In those dependent on infections of the infant itself we invariably find evidences of nutritional disturbances, whether the infection be local, systemic, or confined to the intestinal tract. The clinical picture varies directly with the degree of disturbance of the metabolic function. While, as a rule, the enteral infections are more commonly associated with grave disturbances of the infant's nutrition, it is not uncommon to find the infant severely affected in its ability to meet its nutritional needs by the parenteral infections. While any one of the above enumerated etiological factors may give rise to a marked clinical picture, it is to be remembered that this class of

disturbances in the breast-fed infants are of minor importance as compared with those of the artificially fed (see Nutritional Disturbances in Artificially Fed Infants).

Diagnosis. The diagnosis of the primary seat of infection in the infant is of considerable importance in deciding the method of treatment.

Treatment. Parenteral infections rarely call for restraint in administration of food because of the associated anorexia, and the infant should be nursed (if possible without danger to the mother) directly at her breast.

In the case of enteral infections it may be necessary to withdraw the maternal milk and replace it by a short period of starvation, to be followed by small quantities of breast milk, either taken directly from the breast during short nursings, or it may be best to feed small quantities of expressed milk to the infant at regular intervals.

Not infrequently it becomes necessary to feed these infants by catheter in order to sustain them. And this method of introducing their food should be begun sufficiently early to avoid a catastrophe.

Under no circumstances should they be placed upon food other than the mother's milk when her state of health and the quality of her milk permit.

Inert fluids, such as water, weak tea, broths made from young meats and young fowls, and cereal decoctions should be given between feedings to insure a sufficient intake of water. A careful record should be kept of the twenty-four-hour quantity of all fluids administered, in order to insure the child a sufficient water and food administration.

For conditions in the mother which would justify weaning, see chapter on Weaning and Contraindications to Nursing.

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5. Idiosyncrasy Towards Mother's Milk.

Etiology. This condition is very rare, although it may not be denied that it exists. The etiology and pathogenesis are as yet little understood.

Diagnosis. The diagnosis of this disturbance should be made by exclusion of all other causes that may give rise to a similar symptom-complex. It may be confirmed by the change of the milk either by substituting a wet-nurse or cow's milk for maternal nursing, whereupon the symptoms improve.

The cases in which the mother's milk is totally unfit for the infant are exceptionally rare. More recently considerably more attention has been given to the effect of the mother's diet on the quality and quantity of her milk secretion. The instruction so commonly given to the mother, to the effect that she may eat whatever she likes, has, in the light of more recent investigations, shown need for modification. The effect of the diet of the mother on the milk must be considered under two headings: First, what foods disagree with the individual mother to the extent of affecting the quantity of her milk supply. The mother will be the best judge as to what foods she, herself, finds it desirable to eliminate from her diet because of an undesirable effect upon herself. More important, however, from the standpoint of food idiosyncrasy, is the result following the eating of foods by the mother which she, herself, may relish, but which may have an undesirable effect on the child. It is well known that eggs, some cereals, fish and sea foods, certain meats, chocolate, and even cow's milk proteins may result in a sensitization of the infant when ingested by the mother. The more recent work of O'Keefe[1] demonstrated the frequency of such a sensitization in eczema. He studied forty-one cases of this condition in breast-

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[1] O'Keefe: Eczema in Breast-Fed Babies, Boston Med. and Surg. Jour. August, 1921; 185, No. 6.

fed infants, and 61 per cent. of his cases showed a positive reaction to one of the cow's milk proteins. Forty-one per cent. showed a positive reaction to one of the egg proteins, two cases to oats, and one to wheat. About 20 per cent. of the positive cases showed a response to both milk and egg proteins. Apparent cure in about 20 per cent. followed the omission or limitation in the maternal diet of one or more food proteins to which the infant was sensitive.

CHAPTER VII.

METHODS OF FEEDING PREMATURE INFANTS.

1. Infants Nursing at the Breast.

IN most cases we do not feed the more developed premature infant on the first day. It may be wise, however, to place the infant on the breast two or three times during the last half of the first day, after the circulatory and respiratory functions are well established, so that the infant may become accustomed to nursing. We are now confronted with two important factors, first, the ability of the infant to nurse the breast; and secondly, sufficient and proper development of the nipples to allow of the infant's properly grasping the same. If the infant is sufficiently developed to take hold of a well-formed nipple, it should be placed at the mother's breast regularly at three-hour intervals on the second day, for two- or three- minute periods, even though there is little hope of the breasts secreting at this time. By this means the infant is trained to expect its food at regular periods, and at the same time the maternal breast is stimulated. When a wet-nurse can be supplied in the home who has her own infant with her, the latter can be used to stimulate the breasts of the mother, and the new infant can have one of the wet-nurse's breasts set aside for its use. Where the infant is very weak, the breast set aside for it can be made to secrete more freely by simultaneously placing the wet-nurse's baby on the opposite breast during the period of nursing.

We have found this to be a very valuable expedient. However, with this latter method of procedure the quantity taken by the premature infant must be accurately measured to prevent overfeeding by weighing the in-

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fant before and after the nursing period. Nursing directly from the breast has the added advantage of developing the baby's sucking muscles, preventing contamination of the milk, and stimulating the breasts by the natural method. It should, however, be remembered that a weak infant may nurse the maternal breast for a considerable time, and yet the amount of food taken may be insufficient. This is especially true of that class of infants who are inclined to go to sleep at the breasts. Here, again, weighing is of the utmost importance. When the infant is too weak to nurse sufficiently to satisfy its needs, as ascertained by weighing, the nursing should be followed by substitute feeding with expressed milk, either by the bottle or one of the other methods to be described. These rules do not apply for the first and second day, when only rarely more than four or five meals should be given. In very weak infants, and those subject to regurgitation after taking small quantities of milk, it may be necessary to feed more frequently in periods varying from two to two and a-half hours, as may be indicated by the quantity retained, or better results may be obtained by catheter feeding (to be described later) with four-hour intervals.

2. Infants Too Weak to Nurse the Breasts.

In this class of infants, wherever possible, they should be fed without being removed from their bed or the incubator, if used, so as to avoid all careless exposure of the infant. The cause of inability to nurse may be due to several factors: (1) Infants unable to swallow; this is usually because of improper development of the center in the medulla, or lack of co-ordination on the part of the pharyngeal muscles and tongue. This is usually made evident by the milk flowing from the dependent part of the mouth. In such cases it is generally necessary to resort to catheter feeding. (2) Those too weak to nurse,

and who may appear to be almost dead; in this class there is great danger in handling the infant, and it is best fed in the bed. (3) Those who will not suck. (4) Those vomiting after every feeding. (5) Those becoming cyanotic after feeding. In the latter cases it may even be necessary to resort to such methods as gentle friction, artificial respiration, best performed by gently compressing the thorax, warm baths, oxygen, etc.

Methods. One of the following methods can be selected for feeding these infants:

1. *The nasal spoon,* which can be used either by pouring the milk slowly into the nose or into the mouth. The latter is to be preferred, because of the dangers due to decomposition of the milk in the nose and naso-pharynx, with secondary development of rhinitis and pharyngitis.

2. *A medicine dropper* for mouth feeding. This is possibly one of the best methods for feeding this class of infants, as it is simple of application, and a small dropper is easily obtainable. As in all other methods, the food should be administered very slowly.

3. *Nursing From a Bottle.* For this purpose the small nipples commonly sold on doll nursing-bottles are of the proper size, and can usually be obtained of proper quality. We have not infrequently perforated the rubber end of a medicine dropper and used it for this purpose. The bottle to be used can either be an ordinary 1-ounce or 2-ounce medicine bottle, or, better, the special bottle which was designed by the author for this purpose. This bottle holds 2 ounces of milk, is graduated in cubic centimeters, has a ground glass neck which coapts perfectly with the bulb on the special breast-pump, and which after being filled is corked with a ground glass stopper, and which has the added advantage in that the milk is in no way handled after it leaves the breast.

4. *The Breck Feeder.* This has the added advantage that the milk can be passed into the pharynx without effort on the part of the child when it is too weak to

nurse. This has the one disadvantage of too rapid feeding if not properly controlled.

5. A rather slow but satisfactory method of feeding the infants is by *expressing the milk directly from the nipple into the infant's mouth* during the feeding period.

Fig. 5.—Breck feeder for premature infants.

6. *Catheter Feeding by Mouth* (gavage). For this purpose a small funnel is attached either directly or by means of a short piece of rubber tubing with a glass connection to rubber catheter. A Nelaton catheter is used (best a No. 12 French),[1] about 25 to 40 cm. long (10 to 16 inches), marked in centimeters or inches, so

[1] No. 12 French—No. 8 American—No. 5 English. Diameter—4 mm.

that at all times its position can be estimated. The infant should be fed in the incubator, its crib, or on the dressing table. Its head should be slightly lower than the body. The passage of the catheter is usually effected without difficulty by grasping it as one would a pen, and passing it in the midline to the pharynx, gradually push-
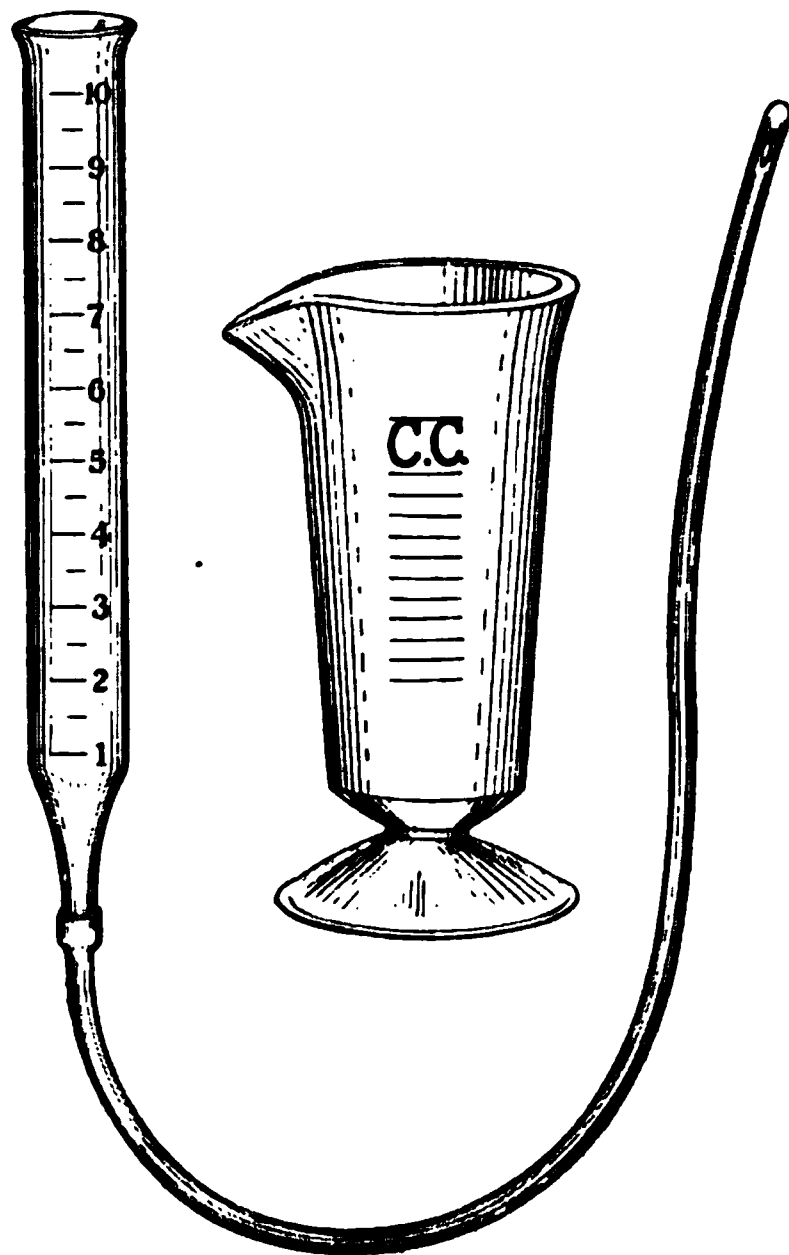

Fig. 6.—Apparatus for gavage and lavage.

ing it into the esophagus. This is usually accomplished without difficulty, because of the poorly developed pharyngeal reflexes, and rarely results in retching or vomiting. In infants who retch during the passage of the catheter, vomiting may be expected because of the fact that these latter infants not infrequently belong to the spasmophilic group. The danger of passing the catheter into the larynx is minimal. It is rarely necessary to pass the catheter more than 10 centimeters (4 inches)

beyond the infant's lips, and we have found it equally as practical to limit the passage of the catheter to 7.5 centimeters (3 inches). In most instances this does not reach the stomach, but has the added advantage of preventing

Fig. 7.—Feeding baby with catheter.

trauma to the cardiac end of the stomach and the gastric mucosa. When a graduated catheter is not at hand, it may be marked at 10 centimeters with indelible ink, and this used as the maximum point for passage. A fairly safe maximum for the passage of the catheter can be ascertained by measuring the distance from the glab-

ella to the epigastrium in the individual infant. The desired quantity of milk is allowed to flow into the stomach, slowly, by raising the funnel only very slightly above the level of the body. After feeding, the catheter is firmly compressed to avoid all leakage into the pharynx, and the catheter then removed, but not too rapidly. The milk to be fed should be measured in a graduated glass, and the latter kept close at hand in order that the amount given can at all times be estimated.

A complete record of every feeding, both as to the time and the amount, should be kept. This is especially important in institutions where the nurses have a number of infants to observe, and is greatly facilitated by a time-clock registering the day, hour, and minute of each feeding. The nurse records the quantity of milk taken, which in breast-fed infants is obtained by weighing the infant both before and after feeding on an accurate scale, or in infants too weak to nurse by measuring the quantity in a graduated glass before feeding.

3. Proper Time for Beginning Regular Feeding.

Due to the tendency toward the rapid development of acute inanition in this class of infants, the greatest danger is that of too long delay in establishing regular feeding. Therefore it is often impossible to wait for the mother's milk to appear. We believe that it is, however, unwise in most instances to attempt to feed with milk during the first twelve to twenty-four hours, rather preferring to allow the circulatory and respiratory organs opportunity for proper accommodation to their new environment. During this time the loss of body fluids through evaporation from the skin and respiratory tract due to the warmth of the incubator, and the excretions through the kidneys and bowels, should be recompensed by the regular administration of water or some other inert fluid.

We endeavor to administer by the tenth day about one-sixth of the body weight of water (inclusive of that contained in the milk if given) in twenty-four hours.

In smaller infants the first milk is given diluted one or two times during the first four days. After the first twenty-four hours water can be administered partly with the food, and otherwise between feedings. If for any reason the water is not well retained when given by mouth, it can, at least in part, be administered by rectum. Example: An infant weighing about 1200 grams should receive 200 mils of water; should this infant receive 50 mils of milk, this can be diluted with 50 mils or more of water or sugar solution, and the remaining 100 mils administered between feedings.

If a stimulant is indicated, a few drops of brandy (6 to 15 in twenty-four hours) may be added to the water or sugar solution during the first twenty-four hours. Half strength of Ringer's solution prepared as follows can be used to very good advantage for rectal adminis-tration:

NaCl	7.5 Gm.
KCl	0.1 "
CaCl	0.2 "
Water	1000.0 mils.

We have made it a rule never to start milk feeding until after the first bowel movement. Not infrequently the removal of meconium may be accomplished by the administration of a small quantity of physiological salt solution through a catheter passed one or two inches into the rectum. This is done to remove the meconium before infection of the intestinal tract through the administration of food. Occasionally it is necessary to administer 5 drops of castor oil to obtain slight purgation.

4. Feeding From the Second to the Tenth Day.

It must be remembered that the general rules as applied to the feeding of premature infants do not hold for the first ten days of life. The early feedings must necessarily be small, and the increases gradual. Two grave dangers present themselves during the first period of the infant's existence: (1) overfeeding and (2) starvation, the latter usually resulting from an inability to supply sufficient quantity of human milk, following an attempt to await the natural secretion of the mother's breast. Overfeeding results either in vomiting or, more seriously, in stomach distention, which leads to asphyxia and cyanosis. Underfeeding in these weak infants soon leads to inanition. From the second day these infants should be fed regularly day and night, every two or, better, three hours, depending upon the infant's condition and the method of food administration. Not infrequently where the quantities taken are very small, ten to twelve feedings are required in twenty-four hours. It may even be necessary in very weak infants to feed minimal quantities every hour.

The question of the number of feedings will be discussed in detail later.

It is practically impossible to formulate definite rules for feeding premature infants during the first ten days, because of their great variation in weight and development. Therefore it becomes necessary to *feed each infant individually*.

During the first days it is often difficult in infants weighing 1000 to 1200 grams or less to feed more than 20 to 50 mils of milk per day, and it may be necessary to limit the food to this quantity during the first ten days. It is our rule to start feedings in this class of cases with a maximum of 4 mils per feeding, not infrequently using one-fourth or one-half human milk at the start, and the balance water.

The feedings should be increased by 1 mil at a time, and with the first evidence of regurgitation the quantity should remain stationary. Even in favorable cases during this time 30 to 50 calories per kilogram is likely to be the maximum that can be fed with impunity.

The small feedings which can be assimilated, and the low energy quotient during the first two or three weeks, must be considered physiological, and as we rarely see an increase in weight with feedings of less than 90 calories per kilogram, we are confronted by a rapid loss in body weight during the first days of life. In favorable cases this is usually followed by a stationary weight, or moderate fluctuations after the first four to seven days. Occasionally an infant is seen in whom there is sufficient water retention to avoid most of the initial loss in weight. One should, therefore, remember that even with frequent feedings with human milk, either at the breast, by hand, or gavage, it is rarely possible to feed more than the minimum requirements without causing vomiting.

5. Feeding After the First Ten Days.

There has been considerable discussion as to the food requirements of premature and underweight infants during the past few years. Budin gives us the rule that premature infants of less than 2500 grams after their tenth day require one-fifth of their body weight (200 Gm. per kilogram of body weight), or 140 calories, while the full-term infant of normal development requires one-seventh of its body weight (140 Gm. per kilogram body weight), or 100 calories per day. On the other hand, Birk believes that the more fully developed premature infant, and those nearing the normal, will thrive on one-sixth to one-seventh of their body weight.

Our opinion, based on a series of experiments made on a number of premature infants, is that they require higher food values, or at least the maximum required by

normal infants, for the following reasons: (1) the greater body surface as compared with the body weight; (2) in the normal infant the requirements decrease with the age, and therefore in the premature the quantity required varies inversely with the fetal age after the first weeks of life; (3) the need for body development is relatively greater in the premature than in the full-term infant; (4) a kilogram of body weight in the fat-poor premature infant cannot be taken as parallel in feeding to the well developed full-term infant, with its preponderance of fatty tissue. This latter point must also be considered in the feeding of the marasmic infant, to obtain a proper gain in weight as compared with the lower requirements in the fat, full-term infant.

6. Number of Feedings Daily.

Our own experience has led us to adopt a conservative position in that we have grouped the infants nursed at the breast or fed from the bottle or by feeders into two general classes: (1) those weighing under 1500 Gm., and (2) those above this figure, based on the tendency of the smaller infants to become exhausted when the feedings are long continued. The former are fed at 2-hour intervals during the day, and 3-hour intervals at night, as follows: 6 A.M., 8 A.M., 10 A.M., 12 M., 2 P.M., 4 P.M., 6 P.M., 9 P.M., 12 P.M., and 3 A.M.—10 feedings during the twenty-four hours. The larger infants are fed on a 3-hour basis, 8 feedings being given during the twenty-four hours. These figures should in no way be construed as arbitrary. All feedings are more or less dependent upon the general development of the infant in relation to its digestion and metabolism, its retention, and upon the larger quantities of food necessarily given to meet its nutritional requirements, and a careful attention to gastric distention, regurgitation, asphyxia, cyanosis, and other respiratory complications.

It has been our personal experience to meet with considerable difficulty in attempting to meet the large food requirements in smaller infants without resorting to catheter feeding. In these we have adopted the longer interval between feedings, of four hours with six feedings in twenty-four hours, the individual meal in catheter feeding being greater in quantity. Notwithstanding the fact that catheter feeding offers little difficulty and few dangers in experienced hands, this may not be true with those not skilled in its use. A considerable number of our cases have, however, thrived satisfactorily on quantities of milk less than one-fifth of their body weight per day, and one should always remember that it is a safe axiom not to force the feeding in these cases as long as their general development is progressing satisfactorily and their weight curve is good.

7. The Amount of Each Feeding.

The statistics as to the stomach capacity for food in premature infants indicate that this varies within considerable limits, even in infants of the same fetal age, as does also their ability to digest and assimilate food. The weight and length, naturally excluding congenital diseases and deformities, will be far more dependable as a guide to stomach capacity than the fetal age. As no definite rules can be established governing the amounts of individual feedings, we begin with what could be considered minimum quantities and gradually increase the amount of feedings as the infant develops an ability to digest it. It is our rule, as previously stated, during the first few days to feed small total quantities varying from 20 to 50 mils of milk per day, dividing these totals by the number of feedings to be administered (eight to ten), thereby feeding from 2 to 6 mils of milk per feeding. The feedings can then be increased by 1 or more mils at a time, and in the absence of vomiting the individual feedings can be increased more or less rapidly

until the weight loss ceases or an increase in weight oc-
curs. Even in favorable cases, weighing over 1500 Gm.,
75 to 150 mils per kilogram weight (50 to 100 calories per
kilogram) is likely to be the maximum that can be fed
with impunity or safety during the first ten days.

8. Daily Gains.

These are not necessarily in proportion to the changing
quantity of milk administered, as many ˙factors, such as
condition of the bowels, quantity of the urine passed,
temperature of the infant's surroundings, will neces-
sarily influence the weight. This is more especially
noticeable in observations continued during a short period
of time. An average greater daily gain than 20 Gm.
is unusual when the infant's food is limited to one-fifth
of its body weight. An average of from 10 to 20 Gm.
daily can in most cases be considered satisfactory.

9. Artificial Feeding.

There can be no comparison between the results to be
expected in feeding premature infants on human milk,
and those to be obtained with artificial food. With
human milk taken from a well regulated department for
wet-nurses the milk can be obtained fresh, practically
sterile; it is more digestible; its constituents are of the
quality and in the proportions required for the growth
and development of the human body; and it is live, and
contains many of the immunity-conferring properties, as
evidenced by the resistance of a breast-fed infant to in-
fections and contagious diseases. Most of these proper-
ties and advantages are lacking in the dead foods used
in artificial feeding. Therefore, if it becomes necessary
to resort to artificial feeding, the selection of the food,
its preparation, and its adaptation to the infant must all
be given the most painstaking consideration. Many
varieties of artificial diet have been suggested by various

authors, such as simple milk dilutions, cream and top-milk mixtures, skim and buttermilk mixtures, malt soup preparations, condensed and evaporated milk, etc. The results with the various diets are to a great degree dependent upon the physician's intimate understanding of and directions for the use of the individual food.

Quantity of Food. It must be remembered that the figures quoted for feeding on breast milk are the maximum that can be assimilated, and in most instances these amounts more than fulfil the immediate needs of the infant's existence, and can be considered (and in most instances would be) excessive quantities for artificial feeding in the first few weeks of life, because of the greater difficulty in the digestion of cow's milk. One hundred calories per kilogram is the maximum quantity that can be digested by most premature infants, and in many instances one must be satisfied with a sustaining diet bordering on 70 to 80 calories, and they must at all times be closely watched for evidence of overfeeding, as it is dangerous to exceed the actual food requirements, and the first evidence of digestive disturbances or of intercurrent infections should lead to the feeding of human milk. During the first days the same rules for minimal feedings must be observed as in feeding with breast milk.

Quality of Food. Opinions vary greatly as to the best food for an artificial diet. Ordinary milk, water and sugar mixtures are rarely well taken. Pfaundler suggests rich fat and low protein milk mixtures; but in this feeding we have seen fat diarrhea resulting. Budin obtained the best results with peptonized boiled milk, using fresh pancreatic extracts for this purpose. Finkelstein, Oberwarth, Birk, Neumann, Von Reuss have obtained their best results through the use of boiled buttermilk mixtures, prepared according to the following formulæ:

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Buttermilk or skim milk .................... 1000
Flour (dextrinized) ...................... 10
Sugar (cane) ........................... 40
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The above being used for the first feedings.

Buttermilk or skim milk 1000
Flour (dextrinized) 15
Sugar (cane) 60
  For later feedings.

Maltose-dextrin compounds can be substituted for the cane-sugar if desirable.

Chymogen or pegnin milk has given us most satisfactory results in the artificial feeding of the premature infants. This latter preparation is little more than a boiled milk in which the curds are precipitated in a fine, flocculent form, about the size of that of human milk, before it is fed to the infant. It is best diluted before use. This preparation should be started with 1 part chymogen milk and 3 parts water, following the directions for increases in quantity and quality as given for human milk. Because of the low carbohydrate content of such mixtures, 0.5 per cent. of lactose should be added after the first few days, and the amount gradually increased to 3 per cent.

When even only insufficient amounts of human milk can be obtained, artificial feeding should be used as a supplement and not as a substitute.

10. Conclusions.

1. The weight, temperature, stools, absence of abdominal distention, cyanosis and well-being of the infant should be the guide for increase in the infant's diet.

2. The utmost care is necessary in increasing the diet of the infant during the first days of life. The gastro-intestinal tract offers the best evidence for increases. Vomiting and abdominal distention and associated cyanosis are the prime indications for stationary or decreased amounts of feeding.

3. An initial weight loss during the first ten days must be considered physiological.

4. These infants, therefore, should be fed small quantities, frequently repeated, every two to three hours during the day and night.

5. After the first twelve hours human milk may be fed diluted with one or two parts of water and sugar, with a caloric value approximating 15 to 30 calories (20 to 40 mils, ⅔ to 1⅓ ounce of human milk to the kilogram of body weight).

6. From the second day on, in the absence of indigestion, the food may be increased by 10 calories daily per kilogram (15 mils daily per kilogram). In the presence of digestive disorders greater care is necessary to maintain the metabolic equilibrium (120 mils, 4 ounces of milk to the kilogram of body weight).

7. It is of the greatest importance to administer a sufficient supply of water to counterbalance the rapid evaporation due to artificially heated and dried air, and the excessive excreta, more especially during the first few days. About one-sixth of the body weight of water, inclusive of that contained in the milk, should be fed in twenty-four hours by the tenth day.

8. It is to be remembered that a standstill in the weight-curve, and indigestion with bad bowel movements, frequently result when 200 mils (140 calories) per kilogram are exceeded.

9. All intestinal disturbances in premature infants should be given the utmost consideration.

10. The method of administration of food in each case varies with the vitality of the infant.

11. In all cases of prematurity, syphilis should be thought of; and in cases in which there is the slightest suspicion, the infant must not be placed directly on the breast of a wet-nurse.

PART III.

Artificial Feeding.

CHAPTER I.

RECENT PROGRESS IN ARTIFICIAL FEEDING.

THE presentation of the subject of artificial feeding without a review of the progress and evolution which our ideas on this subject have undergone during the past years might easily mislead the student to the belief that the last word in artificial feeding of infants has been said. The men who have given this subject the most consideration, we believe, would agree that much is to be hoped for in the future in artificial feeding.

It is most difficult to present in a concise manner the best that we have learned in artificial feeding so that it may be practically applied, because of two very important factors which make for success: (1) a careful interpretation of the needs of the individual infant, and (2) experience on the part of the feeder to meet those needs.

It remained for the American school of pediatrics to do the pioneer work in placing artificial feeding on a scientific basis.

Pepper and Meigs, of Philadelphia, gave us the first rational method in milk modification. They more especially attempted to vary the percentages of casein in cow's milk, believing that the excessive quantity contained in cow's milk was in great part the cause of feeding difficulties. This was accomplished by diluting the milk and adding milk-sugar and cream to make up the deficiency in energy value.

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Rotch, of Boston, made further advances in infant feeding in that he taught us that fat and sugar, as well as protein, were important factors in the disturbances of the artificially fed infants. His work on percentage feeding, whereby he increased or decreased the various constituents of human milk to meet definite clinical pictures, was probably the first epoch-making advance in infant feeding, and his system of feeding has since been known as "the percentage method" of infant feeding.

The German school, of which Rubner and Heubner were the chief advocates, gave us the so-called "caloric method" of feeding, by which they sought to provide the number of heat units required by the infant, basing their estimations on the infant's weight. Of this method we will have occasion to speak later. It is sufficient to state that we do not now use this as a method of feeding, but find a check on the caloric contents of the food of inestimable value in determining the value of our mixtures in avoiding over- and under- feeding. The German school have never attempted the refinements in the percentage composition of their mixtures as advocated by the American school.

More recently Czerny and Finkelstein have taught us the dangers of overfeeding with whole milk, and also its individual ingredients, fat, sugar, and salts, individually and in combination. Their studies have, on the whole, ignored the proteins, in all probability due to the fact that protein disturbances other than those seen in infants suffering from an idiosyncrasy to cow's milk are for the most part limited to infants fed on raw cow's milk, while most of the Continental clinics have for several years fed boiled milk. Their studies and conclusions will be more fully discussed under the disturbances of artificially fed infants.

During the past few years there has been an increased tendency to boil cow's milk before feeding to the infants in American clinics, based on the desire to render the

curd more fragile, and at the same time to destroy the pathogenic bacterial content of the milk. While this has many advantages, it must not be forgotten that it must necessarily cause changes, more especially in the ferments, vitamines, and salts, which are of vital importance to human economy. The ferments are believed to be important to the infant, and this importance has been emphasized especially since the introduction of pasteurization and boiling of milk, for the reason that a high degree of heat destroys them. Some of the ferments are normal constituents of milk, such as lipase, galactase, lactokinase, and diastase. The absence of ferments in the milk indicates that it has been heated. Hamburger's studies on the biologic differences in human and cow's milk are unquestionably of vast importance, and though there has been a tendency in recent years to neglect this factor in infant feeding, we believe that it will again receive more important recognition in the near future. The changes caused in milk by boiling make it necessary to administer fruit and vegetable juices, non-dextrinized cereals, and other foods, such as codliver oil, to prevent the retarded development on the part of the infant.

CHAPTER II.

COW'S MILK.

No method of artificial feeding can perfectly replace nursing or human milk feeding. This must be admitted, notwithstanding the many advances that have been made in infant feeding during recent years.

When breast feeding is impracticable feeding with properly modified milk of other animals is necessary. Although cow's milk shows marked chemical, physical and biological differences from human milk, for practical reasons it has been found to be the one best suited for this purpose.

How Cow's Milk Differs from Maternal Milk. The differences between these two milks summarized in a table which follows are greater than the table indicates. While cow's milk may be modified to approximate woman's milk in composition, it can never be just the same or just as good for infants.

Cow's milk is more opaque than human milk, although the latter may contain a greater percentage of fat. This is due to the opacity of the calcium-casein, which is present in greater proportion in cow's milk. Cow's milk is faintly acid or amphoteric when freshly drawn, but ordinarily is distinctly acid in reaction when consumed. Human milk is amphoteric or alkaline.

There is three times as much protein in cow's milk as in human milk. The reason for this is obvious, when we recall that the ratio of the growth of the calf to that of the infant is about as 2: 1. Furthermore, the protein in cow's milk consists chiefly of casein (3.02 per cent.) and little lactalbumin (0.53 per cent.), while human milk contains 0.59 per cent. of casein and 1.23 per cent. lactalbumin. The sugar in the two milks varies greatly in

amount, but not in kind. Cow's milk contains almost four times the amount of inorganic salts compared to woman's milk. In cow's milk calcium and magnesium are in greater proportion, while in woman's milk potassium and sodium bases are in relatively greater amounts. These differences have an important bearing upon infant's metabolism. There is no great difference in the average amount of fat in the two milks; however, both in human milk and in cow's milk the fat is the most variable constituent.

The curd from cow's milk is usually tougher and in larger masses than in human milk. There are also differences in antibodies, ferments, etc.

Cow's Milk		Human Milk
Amphoteric or acid	Reaction	Amphoteric or alkaline
1.029 to 1.034	Sp. gr.	1.010 to 1.040
3.5 per cent.	Proteins	1.5 to 2.0 per cent.
3.02 per cent.	Caseinogen	0.5 to 0.75 per cent.
0.53 per cent.	Lactalbumin	1.23 per cent.
Clots in large lumpy curds	Effect of rennin	Clots in fine curds
4.0 per cent.	Fat	3.5 to 4.0 per cent.
4.5 per cent.	Lactose	6.0 to 7.0 per cent.
0.75 per cent.	Salts	0.2 per cent.
13 to 14 per cent.	Total solids	12 to 13 per cent.
86 to 87 per cent.	Water	86 to 88 per cent.
Never sterile	Bacterial contents	Practically sterile

Biedert, whose theory found many followers at one time, believed that casein of the cow's milk was the disturbing factor in artificial feeding.

The large, tough curds forming from the casein of raw cow's milk differ considerably from the fine flocculent curds of the human milk casein. Steps have been taken to make the cow's milk curd resemble the human milk curd in its physical properties, such as boiling the milk, citration and addition of cereal waters, and it was found that this modification considerably improved the results of artificial feeding,

The differences in the fat contents of the two milks have less frequently been drawn upon for explanation of frequent nutritional disturbances on artificial feeding, although it has positively been established that fat plays an important part in the nutritional disturbances of the artificially fed infant. The butter prepared from cow's milk contains 10 per cent. of volatile acids, while that prepared from the human milk only 1.5 per cent. And especially the irritant butyric acid glycerid, which is contained in 6 per cent. in butter prepared from cow's milk, is contained only in traces in human milk. The fat drops of cow's milk are also on the whole much larger than those of human milk.

Lactose is the principal sugar in both cow's and human milk, average human milk containing 6 to 7 per cent., and cow's milk 4 to 5 per cent. This increased sugar contents of the human milk, with its fermentation, accounts for the laxative effect of breast milk feeding when the milk is abundant.

L. F. Meyer has experimentally shown that salts of the cow's milk, which vary both quantitatively and qualitatively from those of human milk, have unfavorable influence on children with nutritional disturbances. While we cannot from these experiments conclude that the same holds true for normal, healthy children, yet we have to admit that the salt contents of the two milks are of great importance in artificial feeding.

Escherich and Hamburger were of the opinion that human milk contained ferments which favorably influenced the processes of metabolism. Salge found that tetanus and diphtheria antitoxins could be utilized by the infant only when fed in human milk, while when contained in the milk of other species they did not get into the body fluids of the infant. But whether these biologic differences are of great importance to the infant remains to be proven.

Although it seems probable, yet it has not been demonstrated that cow's milk feeding taxes the digestive functions of the infant's organism more than human milk feeding.

Of great importance is the bacterial contents of the milk, the human milk being either sterile or of low bacterial contents, while cow's milk is never sterile, and not infrequently its bacterial content is very high. Sterilized, pasteurized, and certified milk were the practical results of the efforts to obtain germ-free milk for infant feeding.

The milk for infant feeding must come from healthy cows, must be obtained in clean manner into clean receptacles, must be cooled very soon after milking in order to keep down the bacterial content, and kept cool afterwards. It must be delivered to the consumer as soon as possible in such a way as to prevent any contamination, and must be handled in the home, cleanly, in sterile receptacles, and at all times be kept cool.

The cow from which the milk is obtained must be entirely healthy, and be especially free from tuberculosis and glanders, tuberculin and mallein test being advisable as a routine, besides general examination of the cow. The cows must be kept clean, in a clean stable, which is well ventilated and drained. No dust, manure, or fodder, except that used for immediate feeding, should be kept in the stable. The cows should be kept clean, but even then they should be cleaned again immediately before milking.

The milking must be done in a clean way and milk kept clean afterwards, in order that the bacterial count may be as low as possible. Dry feeding of the cows is preferable, since on this feeding the feces is less liquid, and cows can be kept clean with less difficulty. The milkers should be free from any communicable disease, and be of clean habits. The udders of the cows and the hands of the milker should be scrubbed with warm

water and soap immediately before milking, and anti-septic solution may be applied afterwards. Milking should be done into covered cans, and milk made to pass through a filter first. The cans should be always cleaned immediately after the milk is poured out, first with cold and then with hot water, and also rinsed out with hot water before milking. The first few ounces of milk should be discarded, since this milk contains large amounts of bacteria that are washed out from the excretory ducts.

Cooling the milk after it is obtained is a very important step in the production of clean milk. The milk having been obtained with the above-described precautions, with as few bacteria as possible, should be cooled at once in order to prevent growth and multiplication of the bacteria that have entered the milk in spite of all the precautions. This is accomplished by special cooling apparatuses, or simply by pouring the milk into sterilized bottles, closing with sterilized cap, and putting on ice. The milk in bottles should be kept iced until it reaches the consumer, which should not take longer than twenty-four hours.

In the home precautions should be taken to prevent additional contamination, and to keep the milk iced to prevent further growth of bacteria, until everything necessary is ready for making the proper mixture for infant feeding. Many good milks are spoiled on the door-step of the home between the hour of delivery and placing the milk in the ice-box. All the utensils and vessels used for preparing the mixture must be perfectly clean and sterilized by boiling. As soon as the mixture is made it should be put into the ice-box again and kept there, portions being taken during the day for individual feedings, and warmed separately just before feeding.

Certified Milk. The term "certified milk" was coined by Dr. Henry L. Coit, of Newark, N. J., who in 1892, needing good milk for his own baby, formulated a plan

for the production of clean, fresh, pure milk under the auspices of a medical milk commission. The term "cerr tified milk," then, is the milk of the highest quality, of uniform composition, obtained by cleanly methods from healthy cows, under the special supervision of a medical milk commission.

The use of the term "certified milk" should be limited to milk produced in accordance with the requirements of the American Association of Medical Milk Commissioners. The first requisite in the production of certified milk is to enlist the co-operation of a trustworthy dairyman who enters into a contract with the medical milk commission. In accordance with the terms of this contract, the dairyman binds himself to comply with the specifications set forth, in return his milk is certified.

The dairies are subjected to periodic inspections, and the milk to frequent analyses. The cows producing certified milk must be free from tuberculosis, as shown by the tuberculin test and physical examination by a qualified veterinarian, and from all other communicable disease, and from all diseases and conditions whatsoever likely to deteriorate the milk. They must be housed in clean, properly ventilated stables of sanitary construction, and must be kept clean and properly fed and cared for. All persons who come in contact with the milk must exercise scrupulous cleanliness, and must not harbor the germs of typhoid, tuberculosis, diphtheria, or other infections liable to be conveyed by the milk. Milk must be drawn under all precautions necessary to avoid contamination, and must be immediately cooled, placed in sterilized bottles, and kept at a temperature not exceeding 50° F., until delivered to the consumer. Pure water, as determined by chemical and bacteriological examination, is to be provided for use throughout the dairy farm and the dairy. Certified milk should not contain more than 10,-000 bacteria per cubic centimeter, and should not be more than thirty-six hours old when delivered.

Inspected Milk. This term should be limited to clean, fresh milk from healthy cows, as determined by the tuberculin test and physical examination by a qualified veterinarian. The cows are to be fed, watered, housed, and milked under good conditions, but not necessarily equal to those prescribed in the production of certified milk. Scrupulous cleanliness must be exercised and particular care be taken that persons having communicable diseases do not come into contact with the milk. This milk must be delivered in sterilized containers, and kept at a temperature not exceeding 50° F. until it reaches the consumer. There should be not more than 100,000 bacteria per cubic centimeter of inspected milk. This milk should be pasteurized.

Market Milk. All milk that is not certified or inspected in accordance with the above definitions, and all milk that is of unknown origin, is classed as "market milk," and should be pasteurized.

Frozen Milk. In our own experience we have found that many infants were made ill by feeding of raw frozen milk which has been rapidly thawed, and allowed to stand in a warm room. Not infrequently vomiting and diarrhea result. These symptoms are obviated when the milk is boiled. Pennington and her collaborators found very definite changes in milk after freezing. They found that when the milk is held at a temperature of 0° C. there is proteolysis of the casein, which is primarily of bacterial origin, and proteolysis of the lactalbumin, due primarily to the native enzymes of the milk. The action of these two agents together is more rapid than that of either alone. The bacteria and enzymes may break down the true protein and carry the breaking down through to peptones, even to amino-acids. There is a fermentation of lactose with the formation of lactic acid, which is largely, if not exclusively, due to bacterial action. The fat, so far as can be determined, is not affected except by the action of bacteria.

Mixed Milk Versus Milk of One Cow. It is far better, other things being equal, to use the mixed milk of a herd in preparing a baby's food than the milk of one cow, because if the milk comes from one cow, and the cow is ill in any way, the baby is almost certain to be disturbed, whereas if one or two cows in a herd are ill, the milk from these cows will be so diluted that the baby will probably not notice it. On the other hand, it is, or should be, self-evident that the milk of a healthy cow properly fed and properly cared for, taken in the proper way, and kept under proper conditions, is better than the mixed milk of a herd which is improperly fed, and whose milk is not carefully obtained or carefully taken care of.

Boiling, Sterilization, and Pasteurization. Before entering into a discussion of this subject, it is only fair to state that the general teaching in America of feeding with raw milk has led to the production of safe, clean certified milk in the large communities where so many fatalities were experienced through the feeding of unclean milk. Any methods of handling milk which will in the least interfere with the proper production of clean milk, and lead to the feeling that unclean milk can be made safe for infant feeding by the application of heat or other methods, would be a backward step in infant feeding, and would necessarily cause dire results. While the European countries, as Germany and France, have advocated feeding boiled milk for many years without fear of bad nutritional disturbances due to the changes in the milk, in America feeding with raw milk has until recently been favored. Increased experience with boiled milk, especially by those who have long used raw milk, leads to the growing conviction that boiled milk is more easily digested than raw milk by dyspeptic infants, and hence by the well infants.

While we do not believe that feeding with boiled milk should be advised as a general measure, when it is possible to obtain a good certified milk, and when the latter

is to be placed in the hands of mothers and nurses who can be depended upon to keep the milk clean and wholesome through proper icing and handling, we do believe that when these requirements cannot be met, that it is safer even in well babies to feed a thoroughly sterilized milk, and that this can be done without danger of development of scurvy and rickets, when these feedings are accompanied by the administration of fruit juices, vegetable soups, and purées and codliver oil.

Brennemann suggests that we must answer the following questions before deciding as to whether we should feed raw, pasteurized, or boiled milk:

(1) Does raw milk offer advantages over boiled milk?

(2) Does boiled milk offer advantages over raw milk?

(3) Does pasteurization solve the problem?

(4) Does certified milk solve the problem?

In answer to the first question we must decide whether the changes caused in milk by boiling, such as partial coagulation of lacto-albumin, caramelization of some of the milk-sugar, its action on casein, inhibiting coagulation with rennin, etc., lessen the nutritive value of cow's milk as an infant food. We believe that the sentiment of American, German, and French clinics, in which boiled milk has been used for a long period of time, is on the whole, most favorable to boiled milk, with its lesser dangers.

Constipation has been suggested as an argument against boiling milk. We believe that constipation in the bottle-fed baby is one of the safest earmarks of the well-being of the infant, and that only that constipation which is due to excessive feeding of fat, with too little carbohydrate, and which will be later described, is an exception to this statement. While with raw milk digestive disturbances are frequently seen before sufficient milk is given to properly nourish the infant, this is far less com-

mon with boiled milk; in fact, it has not infrequently. been our experience that we have overfed with boiled milk, because the infant handles it to so much better advantage. In digestive disturbances, with loose stools, it is digested to much better advantage than raw milk, which frequently results in formation of hard casein curds as well as fat curds. The assertion that feeding with boiled milk results in anemia, underdevelopment and rickets, we believe, is not well founded, and these conditions, when present, are due to other causes. Scurvy developing during the course of feeding with boiled milk has never been seen in our experience, except when some of the proprietary infant foods have been fed in conjunction with boiled milk. That under certain conditions scurvy should develop in presence of long-continued feeding with boiled milk alone, is not to be denied. The dangers, however, are very remote, as testified to by the German and French clinicians. When such dangers are feared, they can easily be overcome, as previously suggested, by the feeding of fresh fruit juices and vegetable preparations together with the milk diet.

Does boiled milk offer advantages over raw milk? Boiled milk when properly handled is relatively free from pathogenic micro-organisms, and if the milk, which has been boiled, was clean milk, also from their toxic products.

Boiling in the home has the great advantage over commercial pasteurization in that, if the milk is raw and spoiled before it reaches the home, this can readily be detected by the housewife. While we know that certain pathogenic organisms may develop in the milk without giving evidence of their presence, and cause formation of toxic bodies which are not removed by boiling in the home, the latter process still offers every advantage over commercial pasteurization. Boiling milk in the home will most certainly remove the dangers from infection with tuberculosis, scarlet fever, streptococcus sore throat, ty-

phoid fever, dysentery, and many other milk-borne diseases. The advantages of boiled milk in the presence of indigestion and diarrhea have already been mentioned. The small, flocculent curd of the boiled milk is also rapidly and more easily digested than the large, tough casein curds of the raw milk. The hard bean-like protein curds are never seen in stools of the infant fed on milk which has been thoroughly boiled, although we have occasionally seen them in overfeeding with cow's milk which has been heated by the double boiler process. These latter cases, however, are exceptions.

Larger amounts and more concentrated mixtures of boiled milk can be fed than in feeding with raw milk. This is a distinct advantage in the beginning of the feeding of atrophic infants. This latter advantage is not to be overlooked. While the large percentage of healthy babies will apparently digest equally well raw and boiled milk within therapeutic limits, it will be found that most authors who do not resort to heating milk will, at least in some other way, modify the curd of raw cow's milk, either by simple dilution, by the use of cereal waters or an alkaline, such as lime water or sodium citrate. We agree with Brennemann in his statements that boiling commends itself as an excellent casein modifier, and that it effectually disposes of the majority of bacteriological problems when the milk is properly handled after boiling.

Pasteurization *versus* **Boiling.** Pasteurization was first recommended because of the belief that boiled milk has scorbutic properties, which could not be laid at the door of pasteurized milk. The question of the relationship between boiled milk and scurvy has already been touched upon. Pasteurization in the home is not a very satisfactory process. Commercial pasteurization, even though properly carried out, is too distant from the probable time of consumption of the food to be a safe measure, unless the milk is properly handled after pasteurization. The best argument presented by the advo-

cates of pasteurization is that the milk is essentially a raw milk in so far as its physiological properties are concerned.

Certified Milk *versus* **Boiling.** Clean certified milk, properly handled, both before and after it reaches the home, and where the cost is not prohibitive, when well digested by the individual infant, still remains the ideal food for artificial feeding. When these requirements cannot be met, boiling in the home is the best method for preparation of milk for the infant.

Various Methods of Boiling Milk. In our own work we have resorted in most cases to the heating of the milk in a double boiler. This has several advantages in that the milk is heated in a closed vessel, and has then a less pronounced flavor than when heated in open vessels, and causes but little pellicle formation, unless we have a very thin column of milk. To overcome this latter, we therefore recommend the smallest double boiler which can be obtained, and which will at the same time hold all of the milk which is to be prepared. The milk mixture is put in the inner receptacle, cold, and the water in the outer vessel also cold. The double boiler is then placed on the stove, and allowed to remain until the water in the outer vessel boils for six to eight minutes. While the milk heated in this manner forms a very much finer and softer curd than that of raw milk, it is not as fine as that of milk boiled directly over the flame. However, in most cases, it answers all purposes, and has the advantages above enumerated. In the presence of gastric and intestinal indigestion and allied conditions, the finer curd of the milk boiled directly over the flame may be more suitable; and in exceptional cases, when boiling over the direct flame for three to five minutes does not give the desired result, milk boiled for 30 to 45 minutes over the direct flame will offer further advantages, and this method is worthy of trial for temporary use.

CHAPTER III.

ADAPTATION OF MILK FOR INFANT FEEDING.

FROM the foregoing it may be seen that there is no perfect substitute for human milk in the feeding of the infant, and therefore every effort should be made to assist the mother in the nursing of her infant.

Since all the attempts made to feed an infant on the food not primarily intended for this purpose are attempts at milk adaptation, we necessarily know that no single method can possibly meet the needs of all infants.

And therefore it must be our object, first, to formulate our rules so as to make them safe and adaptable to the feeding of the majority of well babies, leaving the discussion of exceptional and sick babies for further study. It must necessarily go without saying that the food recommended will be excessive for some and inadequate for others. Every organism has its individuality and its fixation coefficient, and every infant makes a different use of the food administered to it. All infants cannot, therefore, be treated according to the same rule.

While many excellent results have been reported with the various methods described for artificial feeding of infants, and some attempt has been made to place feeding on a scientific basis, we believe that we must concede that the methods are all more or less empirical, and the result will be in considerable degree dependent upon the wide range of food tolerance of the healthy infant. The successful physician must depend on the clinical observation of the individual infant for the success of the method of feeding which he is using. Every formula with which we start feeding should be looked upon in the light of an experiment, and the reaction of the infant to this feeding should be carefully studied.

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ing of the very young infant, the size of the individual meal must be greatly restricted over that as recommended for diluted mixtures, so that it will not exceed the caloric requirements of the individual. Budin recommended that all whole milk fed to an infant should first be boiled, which causes the protein to be precipitated in the infant's stomach in the form of a fine curd. This can be further facilitated by the addition of pegnin or chymiogen, which causes the formation of the fine curds before it is fed to the infant, with no recoagulation in the stomach.[1] Alkalinizing milk by the addition of sodium bicarbonate also results in the formation of fine curds. In some forms of vomiting, small quantities of a concentrated food will frequently be found of considerable value. As a routine measure of feeding, whole milk cannot be recommended.

The Percentage Method or System of Feeding. This is frequently spoken of as the American method, or Rotch's method, because of the fact that Rotch, of Boston, did much to popularize and systematize this method of feeding. Not only did he work out a system of formulæ adapted to infants of varying ages and development, but he also was the means of establishing the first so-called public milk laboratory. The chief objections to this method, as originally described by Rotch, were its lack of flexibility and the difficulty of remembering the various formulæ and their preparation. The followers of the Rotch school state that the percentage feeding, so-called, is not a method of feeding, but merely a method of calculation, and a means of obtaining relative accuracy in the preparation of infants' foods. They have simplified the method as originally applied, lengthened the feeding intervals, still retaining some of the original ideas. It has to a large extent been replaced by the simpler methods of milk modification.

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[1] Brennemann, Archives of Pediatrics, 1917, 34, 81.

Top Milk Feeding. In this method a definite number of ounces of the upper part of the milk, which has stood for a number of hours, is used as the basis for preparing the mixture to be fed.

To successfully carry out top milk feeding, the percentages of fat at various levels in 32 ounces (quart) of milk containing 4 per cent. of fat, and which has stood for six hours or longer, must be known:

Upper 16 oz. has 7 per cent. fat.
" 20 " " 6 " " "
" 24 " " 5 " " "

(1) This method endeavors to provide ample caloric values. In this respect the method may be regarded as successful. (2) There is the idea that casein is not very digestible, and that it is advantageous to feed casein in small quantities, making up the shortage in energy value of the mixture with fat. In the light of our present knowledge, however, we know that the casein of boiled or alkalinized milk, or when mechanically divided by the addition of cereals, is easily digested. (3) The attempt to produce a formula with the percentage of fat in the same proportion as is found in human milk, as well as larger amounts, which, however, frequently leads to fat indigestion, because of the greater difficulty experienced by many infants in handling large quantities of cow's milk fat. (4) The importance of the sugar and salt content of the mixture is underestimated.

This method of feeding, nevertheless, has many advocates, and we would advise that the above shortcomings of the method as originally described be given full consideration by those adopting this method of feeding.

CHAPTER IV.

MILK DILUTIONS WITH THE ADDITION OF CARBOHYDRATES.

IT has been our experience that about 90 per cent. of the infants that come under our observation for artificial feeding will tolerate a wide range of quantitative values in the components of the milk, *i.e.*, fats, proteins, carbohydrates, and salts. And the simpler the first formula on which the baby is started, the easier we find it to meet its later needs for growth and development, by increasing or decreasing the individual elements in the diet. The first step of this method consists in the dilution of whole milk with water, thereby reducing all the ingredients of the milk. When we compare such a dilution with human milk we find that when protein approximates that contained in breast milk, the fat is considerably reduced below that contained in the latter. This in practical feeding we find to be an advantage rather than a disadvantage, and if there be an indication for increasing the fat content of the formula this is easily accomplished by the addition of cream, or top milk, which is, however, usually not necessary, as the deficiency in fat can usually be successfully compensated by adding sugar and starch to the formula. As a result of dilution, the salts, which are about three times as great in quantity in cow's milk, are reduced to more nearly the amounts contained in breast milk. We must, however, remember that there are qualitative differences in the *salt content* of the cow's milk dilution and human milk (page 144).

Feeding should primarily be formulated to promote normal growth and development, to supply energy for the body functions, to prevent disease; and, although of no lesser importance, feeding in disease should be given a secondary consideration in the study of this subject.

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The food must be given in such form that the infant may be able to digest it easily, to assimilate it, and to utilize its constituents for the purposes enumerated above.

The following factors must be considered before estimating the composition and quantity of food for infant feeding.

1. The clinical aspects—that is, the general well-being of the infant—must be given equal importance with the percentage and energy value of the food administered.

2. Is there a normal gain in weight which an infant must show as a sign of full health?

3. The qualitative and quantitative chemical composition of the food, the number of calories available from the total administered, and the proportion of the total fixated in the body must be taken into calculation.

The normal artificially fed infant should manifest the same clinical evidences of good health and progress as are seen in the breast-fed infant. It should be comfortable, which he manifests in a happy disposition. He should be a good sleeper, and awaken regularly for his feedings, and there should be no more occasion for his crying than in the case of the breast-fed baby. His temperature should show maximum excursions of 1° to 2° F. daily. He should have large quantities of subcutaneous fat, and his muscular tissue should be well developed. The turgor of his tissues should be normal. The latter can be estimated by the eye and by palpation. The muscles may be taken between the fingers, and their firmness or softness estimated in this way. By raising a fold of the skin we may determine whether the panniculus adiposus is well developed. The stools, which of necessity must vary with the diet, are firmer and drier and much paler than those of the breast-fed infant, and he should pass one or two daily. Except in the presence of large amounts of carbohydrates, and more especially malt

sugars, they are alkaline in reaction, and have a foul odor.

Therefore, we see that the criterion of good health for the artificially fed infant depends on many things, which together make up the condition of the infant. And we again desire to emphasize that the impression of the general well-being of the infant is a much safer method of estimating its progress than a study of his weight-curve alone.
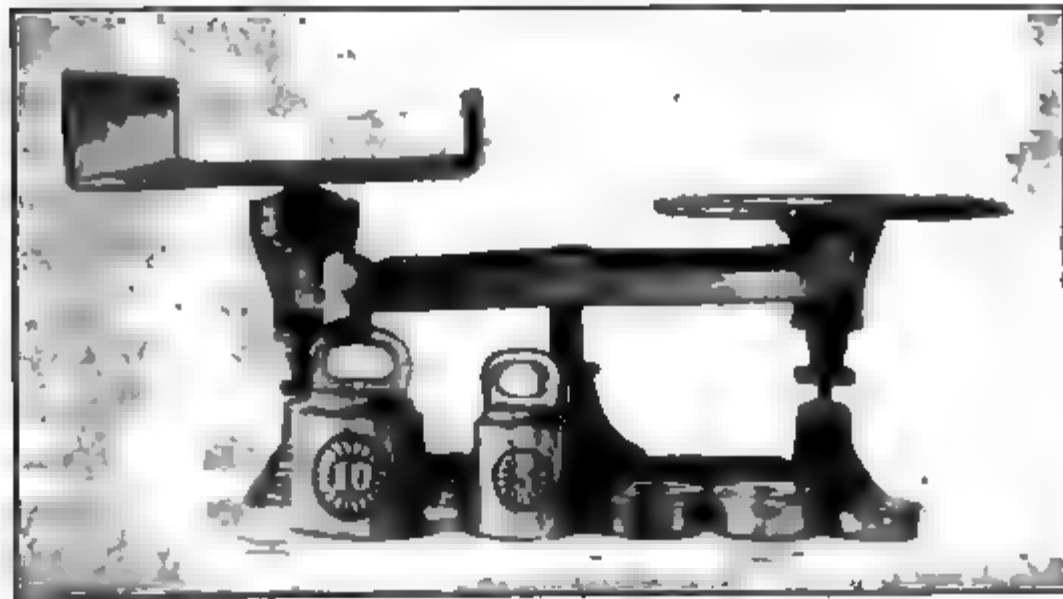
Fig. 8.—Scale for weighing infants.

We have learned to recognize the study of the infant's *weight* as one of the simplest and most reliable clinical factors in estimation of the infant's progress. And while of necessity the diet of different infants necessary to normal weight increases must vary within very considerable limits, the scale offers information which is of inestimable value.

The following may be taken as working averages for comparative purposes, and the estimation of over- and under- weight in infants coming under observation.

Average *weight at birth* 7 pounds (3200 Gm.)

Average *initial loss* 10 ounces (300 Gm.) or about one-tenth of the body weight at birth.

Birth weight regained usually by the fourteenth day.

Weight is *doubled* at the end of the fifth month.

Trebled at the end of the first year.

Average *weekly gain* during the first five months should approximate 5 ounces (150 Gm.), during the remainder of first year 4 ounces (120 Gm.).

Yearly gain during the *second year* 6 pounds (2727 Gm.).

Gain during the *third year* 4.5 pounds (2000 Gm.).

Gain from the *fourth* to the *eighth* year, 4 pounds annually (1800 Gm.).

Gain from the *eighth* to the *eleventh* year, 6 pounds annually (2700 Gm.).

An accurate scale is a necessary equipment for proper infant feeding. Parents should be encouraged to purchase a balance scale with a large scoop.

However, it is not sufficient to base the determination of the amount of food on the weight of the baby alone, since two infants of the same weight may have decidedly different nutritional requirements, dependent upon various factors. The fat baby requires less food per pound than the thin baby—the overfed less than the underfed infant; and the sick baby must of necessity be fed within its limits of tolerance during the acute part of its illness, and the body losses must be compensated by increases in the diet beyond those which we have learned to consider as the normal feedings per pound body weight, as its tolerance for food permits during convalescence.

A healthy infant should, therefore, show a regular gain within certain limitations. It is not absolutely necessary for an infant to add to its body weight every day, as daily irregularities are rather the rule than the exception. The relation of the time of weighing to the feeding, defecation, and urination are factors which must always be taken into consideration. Therefore under normal conditions it is sufficient to *weigh the infant once a week*. It is especially wise to impress this upon a nervous mother.

Further, we must not forget that the weight curve of the nursing infant and that of the artificially fed infant differ widely, so that they cannot be compared directly.

The artificially fed infant, although in the beginning gaining less than the breast-fed infant, in the course of a year reaches the same weight as the breast-fed infant, who at first showed larger gains, but later lagged somewhat in its gains. Much more important than the weight itself is the rising series of successive weight figures.

The clinical aspects, that is, *the general well-being of the infant must be given equal importance with the percentage and energy value of the formula.* In a consideration of the latter two important factors in successful feeding, *the chemical composition must be considered of equal importance with the caloric value.* Otherwise one meets with profound disturbances due to feeding of insufficient or excessive amounts of the components of the diet, difficult of interpretation.

It may therefore be stated that the infant must be fed amounts of fat, protein, carbohydrates, and salts and water suitable to its constitution, age, and physical development, and that these ingredients should be in proper proportion and of sufficient quantity to meet the caloric requirements of its tissues for growth and development. Again, we must not overlook the fact that the constituents of the diet must be in such form as to allow of normal digestion and assimilation.

We have spoken of the wide range of tolerance of infants to their foods, and have mentioned that this, in all probability, accounts to a very great degree for the fact that so many men have been successful in the feeding of infants on a variety of mixtures which varied greatly both quantitatively and qualitatively. There is in all probability another factor which is important in explaining these successes, namely, the fact that to a certain extent fats, carbohydrates, and proteins are interchangeable in their metabolic functions.

Proteins. After passing through the intestinal wall proteins have three functions to perform: (1) to replace used protein (lost through urine, sweat, digestive juices,

cell destruction, etc.) ; (2) to satisfy cell growth, which would be impossible without proteins; (3) to furnish fuel for part of the dynamic loss (fats and carbohydrates are the natural fuel, the protein combustion being incidental only).

There is three times as much protein in cow's milk as in human milk. The reason for this is obvious, when we recall that the ratio of the growth of the calf to that of the infant is about as 2 to 1. Furthermore, the protein in cow's milk consists chiefly of casein (3.02 per cent.) and little lactalbumin (0.53 per cent.), while human milk contains 0.59 per cent. of casein and 1.23 per cent. of lactalbumin.

The proteins are characterized by containing nitrogen. If the nitrogen is determined in the food eaten during the period of the experiment, it is evident that a balance may be struck which will determine whether the body is receiving in the food as much protein nitrogen as it is metabolizing and eliminating in the excreta. If there is a plus balance in favor of the food, it is evident that the body is laying on or storing protein, while if the balance is minus, the body must be losing protein. During the period of growth, in convalescence, etc., the body does store protein, and under these conditions the balance is in favor of the food nitrogen.

It is important also to bear in mind that nitrogen or protein equilibrium may be established at different levels in order to explain the good feeding results with what may be an excessive protein diet. That is, an infant who has been receiving 1.5 Gm. of protein per Kg., and who has excreted the greater part thereof, retaining only such portion as is needed for the body growth, will, upon being fed larger quantities, retain only a similar amount for body growth, excreting the difference in the urine, sweat, and feces. The true cell life does not depend on what has been ingested, absorbed and temporarily fixated, to be eliminated soon afterwards, but on the constant and

stable fixation. The body may become adapted to over-feeding and overfixation, but this is usually of only a short duration, and the excretion of the oversupply is never long delayed. Experimentally, it is found that there is a certain low limit of protein which just suffices to maintain nitrogen equilibrium. Rubner found that when 5 per cent. of the total energy intake was in protein that it was sufficient for maintenance, and that even 4 per cent. was sufficient to supply its actual need when amply supplied with carbohydrate. However, 7 per cent. was necessary to keep up the normal growth.

Examination of the dietaries of civilized races shows that, on the average, 100 to 120 Gm. of protein are used daily by an adult man. A variable portion of this amount passes into feces in undigested form, but we may assume that about 100 to 105 Gm. are absorbed, and actually metabolized in the body. If we take into account the weight of the body, this amount of protein may be esti-mated as equivalent in round number to 1.5 Gm. of pro-tein, or 0.23 Gm. nitrogen, per kilogram of body weight. Chittenden believes that the daily quota of protein per kilogram of body weight may be reduced to one-half this quantity, from 1.5 Gm. to 0.75 Gm. of protein, or 0.12 Gm. of nitrogen, per kilogram body weight.

If the body can be kept in good condition upon 0.75 Gm. per kilogram per day, will an ingestion of more than this (say twice as much) prove injurious or beneficial or indifferent to the body? The full and satisfactory answer to this question must be deferred until more ex-perience is obtained. The newer conceptions in regard to the digestion and nutritive history of the protein foods seem to favor the adoption of a low protein diet. Mankind, when left to the guidance of the natural appe-tites, has always, when possible, adopted the high pro-tein level of 90 to 100 Gm. per day. That mankind has made a mistake in adopting the higher protein level can hardly be claimed on the basis of our present knowledge.

The chief demands for protein are to compensate for wear and tear, and to provide for growth.

Sugars and starches, when added to a diet sufficient to meet an infant's needs, will, temporarily at least, cause a greater nitrogen retention. Fats have little or no such influence. Nitrogen to be retained must be built up into living protoplasm, and to accomplish this salts must be available. Unless they are present, the nitrogen is again excreted. Approximately 1.7 Gm. of ash are retained for each 1 Gm. of nitrogen (Howland), or 0.3 Gm. of ash for each 1 Gm. of protein.

Hoobler believes that the protein needs of the infant are supplied when 7 per cent. of its caloric needs is furnished in protein calories, and states that three-fourths of an ounce of whole or skim milk, or 1.3 Gm. of protein per kilogram (0.6 Gm. per pound) body weight is sufficient to meet these needs. To make up the deficiency in the caloric needs, he adds for each ounce of whole milk one-third of an ounce of sugar or cereal.

Rubner was able to promote normal growth when 0.7 per cent. of the total energy intake was in proteins.

Cowie finds the protein requirement in a two- to twelve-months infant to average 2.4 Gm. per kilogram (1.1 Gm. per pound).

Dunn states that 1.0 Gm. to 1.5 Gm. of protein daily per kilogram (.45-.7 Gm. per pound) of body weight is necessary for the normal infant.

Camerer states the following requirements for each kilogram of body weight in a child between 2 and 4 years of age: proteins, 3.6 Gm.; fat, 3.1 Gm.; carbohydrates, 9.2 Gm.; and water, 75.3 Gm.

It has been our custom to feed approximately 1.5 ounces of milk to a pound of body weight to the healthy normal infant, which would represent 1.5 Gm. of protein per pound of body weight. (3.3 Gm. per kilogram.)

Notwithstanding what has been said on theoretical and experimental studies of the protein needs of the arti-

ficially fed infant as compared with the amount of protein as received by the breast-fed infant, it must be granted that casein, the chief protein of cow's milk, as given in ordinary dilutions to the infants is sufficient to cover entirely the protein needs of the infant, and that its excess rarely causes nutritional disturbances when the tendency to large curd formation is prevented by boiling or alkalinizing the milk.

We have therefore continued to use the protein as contained in 1.5 ounces of milk per each pound of body weight of the normal infant, and in the underfed we have not hesitated to increase this quantity to an amount equal to 2 or even 2.5 ounces per pound, thereby approximating 1.5 ounces per pound of what the baby should weigh for its age. Increases of milk in the diet must be gradual, the additions being guided by the child's ability to handle the food. From what has been stated, it may be inferred that it is wise to establish the protein content in a diet which may then be supplemented by fats, carbohydrates, and salts, because protein is the tissue builder and must necessarily be a basic constituent of all diets.

Fats. Fats are necessary to normal growth and nutrition of the human body. But they to a greater extent than the other food elements can be replaced by proteins and sugars, more especially the latter. This explains the fact that infants fed on low fat mixtures, more especially proprietary foods, such as condensed milk, will continue to gain in weight. However, such development cannot be considered as normal.

Fats furnish part of the heat energy necessary to maintain the body temperature. They are stored as a reserve food. The fat is a protein saver, and when supplied in proper amount but little protein is used for the production of animal heat, thereby allowing for greater protein retention for the growth of the body tissues.

Under normal conditions, the average infant will digest from 2 to 3.5 per cent. of fats. However, some infants

digest fat badly, and when a fat intolerance is once established it is overcome only with great difficulty. In such cases it is necessary to throw the burden of furnishing the extra food necessary on the carbohydrates; and carbohydrates in large quantities are unsafe food for the infant. Such a catastrophe should be avoided, as infants receiving an insufficient amount of fat rarely thrive satisfactorily. We should therefore aim to stay within safe limits. And it has been our experience that most infants will thrive well on the amount of fat furnished by the use of 1.5 to 2.0 ounces of whole milk per pound body weight. When moderate quantities of fat are fed, we avoid the acute clinical picture of fat overfeeding associated with vomiting and diarrhea, and not infrequently a high temperature, and occasionally convulsions. On the other hand, the moderate quantity of fat contained in the diet necessitates a high percentage of carbohydrate feeding, which in turn avoids the so-called fat-soap stools, with their tendency to rob the body of an excessive amount of calcium and magnesium. For the formation of a fat-soap stool it is necessary that we have an insufficiency of carbohydrates and a relative excess of proteins, as putrefaction is necessary for the production of these stools, while fermentation opposes their formation. And in the presence of excessive fermentation the putrefaction is limited.

It may therefore be stated that while the tolerance for fat of cow's milk varies greatly in different individuals, most infants, however, will digest and assimilate 1.5 to 2.0 Gm. of fat per pound body weight daily, (3.3-4.4 Gm. per kilogram) which is the quantity represented in 1.25 to 2.00 ounces of average cow's milk. This quantity will also supply the body needs for growth and development, when associated with a sufficient carbohydrate.

Carbohydrates. They are used chiefly to supply heat and energy, to supply in part material for fat foundation, thereby replacing in part the fat waste. Because

of their high caloric value they supply a large amount of energy. They are efficient sparers of protein, and will supply energy in case of fat insufficiency in the diet. Synthetically, they are converted into glycogen in the body. Fat is formed from sugar by the subcutaneous cells, which are especially adapted to this function. Sugar is reduced to CO_2 and water, which may be measured by the respiratory metabolism. Normally, sugar is absorbed from the small intestine in greater part, and is not found in the feces. If absorbed in sufficient quantity, they will cause a rapid increase in weight. When insufficient carbohydrate is supplied to the body, it is obtained by breaking down the body protein.

In general, infants have a very high carbohydrate tolerance—much higher than the adult—and even infants suffering from certain forms of nutritional disturbance may retain their ability to metabolize sugar, even though it may have been reduced for fat and proteins. Some infants do not handle sugar well, and among these certain forms of gastro-intestinal disturbances, eczema, etc., are of frequent occurrence.

During recent years much has been written on the superiority of one form of carbohydrate over the other. We can practically exclude the monosaccharides in the consideration of the subject, and speak only of the disaccharides, of which lactose, saccharose (cane-sugar), and maltose are the ones used in infant feeding, of the polysaccharides, as represented by the cereal flours and dextrin, and last, of the mixture of disaccharides and polysaccharides, together with other substances, these mixtures being represented by the various infant foods on the market.

Cane and Milk Sugars. Of recent years there has been a considerable discussion on the comparative nutritive value of milk-sugar (lactose) and cane-sugar (saccharose). In our own experience we have found little to recommend one over the other in so far as their

nutritive value and the limit of tolerance is concerned, except as we have seen a laxative effect from the use of lactose, which is usually not present with the same quantities by weight of saccharose. This is, however, not seen in all infants. Cane sugar will answer the needs of most infants. For practical purposes the following quantities of sugar in addition to that contained in the milk will meet the carbohydrate requirements.

1. Normal full weight infants one-tenth ounce by weight of sugar for each pound of body weight (3 grams for each pound).

One and one-half ounces of milk contain 2 Gm. making a total of 5 Gm. to the pound or 11 to the kilogram. Holt[1] and Fales found that nursing infants took on the average 12 Gm. carbohydrate per kilogram daily; artificially fed infants somewhat more than this. They suggest that an infant of average activity at one year be allowed about 12 Gm. per kilogram decreasing the amount to about 10 Gm. at 6 years.

Carbohydrate needs beyond that furnished by one and one-half ounces of sugar should be supplied by well cooked cereal waters, because of the danger of sugar indigestion. (See mixed diet, page 145.)

The total carbohydrates (sugar contained in the milk, sugar added to the milk, and cereal, if used), should average from one-eighth to one-fifth ounces, (4 to 6 grams) per pound body weight a day. One and one-half ounces of milk, averaging 4.5 per cent. carbohydrate furnishes 2 grams of lactose.

2. In underweight infants the amount of sugar to be added must frequently be calculated on the basis of the normal average weight of the healthy infant of the same age. (See page 143.)

Precautions to be Heeded in the Addition of Carbohydrates to the Infant's Diet:

1. Infants who have been on a low sugar diet should

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[1] Holt, L. E. and Fales, H L.: Am. J. Dis. of Children; xxiv, 44, 1922.

be accustomed to the change by gradual increase of the sugar content of their food.

2. In underweight infants the amount of sugar to start with should be calculated on the basis of their present weight. The quantity of sugar needed for a full weight infant of the same age should then be approximated as rapidly as the sugar tolerance permits.

3. In changing from one kind of sugar to another, it is always a safe rule to reduce the quantity for a few days, further increases being governed by the infant's tolerance.

4. It should be remembered that in some infants the disaccharides when fed in full amounts are liable to produce digestive disturbances. In these infants the sugar in part at least must be replaced by polysaccharides in the form of cereal flours and cereals.

Maltose and Dextrin Compounds can frequently be added to the diet to advantage in the presence of stationary weight, because they can be added to the mixture in quantities approximating one-eighth of an ounce (4 grams) for every pound normal weight, when indicated. It must, however, be remembered that their action on the bowels varies greatly depending upon their maltose, dextrin and alkali content. Thus we find that those of the proprietary foods containing a considerable percentage of dextrin, in the absence of the potassium salt, are constipating (Mead's dextrimaltose No. 1 and No. 2), while those with a higher maltose content together with potassium carbonate (Borcherdt's dri malt soup and Mead's Dextrimaltose No. 3) or with potassium bicarbonate (Mellin's Food and Horlick's Malt Food) are laxative.

Cereal Flours. They can be added to the diet of most infants early in life in quantities varying from 0.5 to 1.0 Gm. ($\frac{1}{60}$ to $\frac{1}{30}$ ounce) of flour for each pound of body weight. Such an addition to the food frequently results in rapid weight increases, and general

improvement of the infant. In older infants, cooked cereals may be used in place of the starch solutions. We have reason to believe from clinical experience that the flours made from unheated cereals have a decided advantage over the dextrinized flours on the market. Whether this is due to vitamines and vegetable proteins contained in the former or to some other distinctive property we are unable to state. The cereals also have a decided influence on the calcium and magnesium balance. The cereals cause retention of these salts, which may have a favorable influence on the weight.

Salts. Salts are necessary in digestion, and in every step of metabolism, from absorption to excretion and secretion. The rôle of salts in both normal and pathological conditions has been given constantly increasing importance in the last few years.

Human milk contains 0.2 Gm. of ash in 100 mils, and cow's milk 0.75 Gm. of ash in 100 mils. The difference in percentage in the human and in the cow's milk is equalized by the body using only what is necessary for its life and growth. The salts are absolutely necessary for the life of the organism.

All the salts except those of iron are found in larger amounts in whole cow's milk than in human milk. In general, cow's milk contains relatively a very large amount of calcium phosphate, while the proportion of iron in cow's milk as compared with human milk is relatively small. There is a great difference in the form in which phosphorus is present in human and in cow's milk. In human milk three-quarters of the phosphorus is in organic combination, while in cow's milk only one-quarter is in organic combination. The iron in neither human milk nor in cow's milk is sufficient to meet the demands in the first year of life; the infant must in part depend on the iron stored during fetal life.

The following table gives the percentages of different salts in 100 parts of ash of human and cow's milk.

	CaO	MgO	P_2O_5	Na_2O	K_2O	Cl	Fe
Human milk	23.3	3.7	16.6	8.0	28.3	16.5	.00015 [1]
Cow's milk	23.5	2.8	26.5	7.2	24.9	13.6	.00007 [1]

Grams of salts per 100 c.c., of milk.

	CaO	MgO	P_2O_5	Na_2O	K_2O	Cl	Fe
Human milk ..	.0458	.0074	.0345	.0132	.0609	.0358	.00017 [1]
Cow's milk	.172	.02	.2437	.0465	.1885	.082	.00007 [2]

In all the constituents except P_2O_5 and iron, the percentages of the different salts in the two milks are practically the same. The higher proportion of phosphorus in cow's milk is due to the large amount of the casein. Though the proportions of the different salts of the ash in cow's milk are so nearly those of human milk, the amount in cow's milk is about three and a half times as great. Unless, therefore, cow's milk has been diluted with more than twice its volume, the amount of these inorganic constituents furnished to the infant is equal to that which he receives in human milk (Holt). Human milk contains about twice as much iron as cow's milk, and dilution of cow's milk results in a decrease in the iron content, which must not be carried too far unless supplemented by other iron-containing food.

The infant receiving undiluted cow's milk, with its greater salt content, lives on a higher plane of mineral metabolism than does the one receiving the breast milk. He absorbs 60 per cent. of the total ash, and retains only about 15 per cent., while the breast-fed infant utilizes to the full his opportunities, and absorbs 80 per cent. of the ash, and retains 40 to 50 per cent. In the majority of infants this excessive salt intake undoubtedly does no harm; the surplus is not absorbed, or is merely eliminated.

"Sodium and potassium are usually well retained, unless severe diarrhea is present, or there is an excess of fat or of sugar in the diet. Under such circumstances

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[1] Holt : Amer. Jour. of Dis. of Child., Vol. x, 1915.

[2] Langstein-Meyer: Weisbaden, Verlag von J. F. Bergman, 1914, p. 22.

they are lost, and the loss is badly borne, and cannot indefinitely be continued. When all available alkalies have been drawn on, the infant breaks down his own tissue to furnish more of these substances, which is an explanation, for a part at least, of the excessive nitrogen excretion under such conditions. When diarrhea ceases, and the intake is sufficient, a positive balance is rapidly instituted.

"The metabolism of calcium has been largely studied, on account of its close relationship to rickets and tetany. Calcium is so largely excreted by the bowel that it is impossible to say how much is absorbed, plays part in the organism, and is then excreted by the intestine and urine, either because it is in excess, or because (as in the case of rickets) the body cannot utilize it. This is also true of magnesium, and to a much less extent of sodium and potassium" (Howland).

The salts are necessary for building up of the body tissue, and each gram of protein retained and built into body tissue requires approximately one-third of a gram of ash.

Water. The quantity of water necessary for the infant is not only of theoretical, but also of vast practical importance. There are many breast-fed infants who obtain a food which is very rich in other nutritive substances, but contains only a small amount of water. These infants may not gain well in weight unless water is added. And, besides, in sick infants it is occasionally necessary to feed them (especially in cases of vomiting, anorexia, infections) with concentrated food, and in these cases the total water intake necessary must not be lost sight of.

The lack of or inadequacy of water is much more dangerous to the infant than a corresponding deficiency in the food.

Water to be Added. The most important fact to remember is that young infants require a minimum of one-

fifth of their body weight in water daily (3 ounces per pound) and in their later months at least one sixth of their body weight (2½ ounces per pound).

Water may be given twice daily from a bottle in quantity sufficient to meet the infant's requirements by feeding the difference between one-fifth of the body weight and the milk mixture. (A ten pound baby should get 30 ounces of fluid. If receiving 25 ounces of milk mixture, a daily total of 5 ounces of water may be given from a bottle between feedings.)

If the infant shows a desire for larger individual meals than are furnished by following the amounts outlined, the total fluids (milk and diluent) can be figured on the basis of 3 ounces to the pound and the additional water required to meet the total day's fluid may be added to the mixture in place of being given between meals.

Estimation of the Caloric Contents of the Food as a Check on Over- and Under- feeding. Calorimetric estimations of the diet must be considered only as a check on under- and over- feeding, and not as a method of feeding. In the infant whose diet usually consists of milk or its constituents and sugar and cereal flours, this is a very simple matter. It should, however, be remembered that there are considerable variations in the caloric requirements of normal babies. The fat and well-nourished infant will require less food to maintain its body heat than the emaciated one. The sick baby will rarely be able to digest its full needs as estimated by its body weight. Therefore as in every other phase of infant feeding, the individual infant must be given primary consideration. It must be remembered that the nutrition of the baby depends upon the quantity of the food assimilated, and not upon the quantity ingested. Less food is being absorbed and utilized in the infant with a deficient power of digestion, and overfeeding will retard the infant's progress. A comparative estimate of the infant's diet, with a theoretical minimum, is of special

value in cases of doubt as to whether the retarded progress is due to insufficient food or defective digestion and assimilation.

Under this system the physician reckons the minimum daily caloric requirements, either from the present weight of the baby or what it should weigh in health, and then chooses the food necessary to meet this requirement, bearing in mind that the fat, carbohydrate and protein contents of the diet must not only meet the caloric requirements, but also be properly proportioned, so as to contain the proper number of grams of each of the constituents to meet the infant's needs for growth and development.

Heubner and Rubner gave us the first definite estimates as to the caloric needs. They found that the average healthy infant after birth requires on the average 100 calories per kilogram body weight, from six months to the end of the first year—approximately 85 calories per kilogram body weight—and that 70 calories per kilogram body weight is the energy quotient on which a baby would maintain a weight equilibrium.

Dunn places this minimum caloric requirement for artificially fed infants as follows:

Birth to 6 months ... 120 cal. per Kg. (55 cal. per pound)
6 to 12 months ...... 100 "   "   " (45 "   "   ")
12 to 24 months ..... 90 "   "   " (40 "   "   ")

Dennett[1] gives the following figures:

Fat infants over 4 months of age .. 40 to 45 cal. per pound
Average infants under 4 months of
  age and moderately thin infants of
  any age .... 50 " 55 "   "   "
Emaciated infants (varying with the
  degree of emaciation) .... 60 " 65 "   "   "

[1] Infant Feeding, J. B. Lippincott Co., Philadelphia, page 58.

Brady[1] gives the following figures as his experience with institutional children: 50 to 55 calories for each pound during the first 6 to 8 months of life.

Our own experience coincides with those of Dennett and Brady in that we find that the figures of Heubner do not meet the requirements of any except the well-nourished infants. Underfed infants not suffering from decomposition (marasmus) must be fed food of a higher caloric value per pound body weight than the normal infants, and while such infants must be fed minimal quantities when first seen, for a proper gain in weight their normal weight must be estimated and their diet gradually approximated to the needs of the weight that they should normally have.

Average infants under 2 months of age ..	30 to 45 cal. per ℔	(65 to 100 per Kg.)
Average infants over 2 months of age ..	45 " 55 " " "	(100 " 120 " ")
Premature and thin infants under 2 months of age	50 to 65 cal. per ℔	(110 to 140 per Kg.)
Thin infants older than 2 months, depending upon their general condition .	55 " 70 " " "	(120 " 150 " ")

During the first few weeks of life of the artificially fed infant it is usually difficult to approximate these figures (see page 151).

Increases in quantity of food should always be gradual, especially in the presence of malnutrition, and the infant carefully observed, and increases made only as the tolerance for food permits.

Estimation of the caloric contents of the food is *not a feeding method* and should be used only as a check on over- and under- feeding, the scale, stool, and general

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[1] J. M. Brady, Institutional Care of Infants, Archives of Ped., 1917, 34, 356.

condition, and particularly the disposition of the infant, being the ultimate guide for dietetic changes.

Energy quotient is the number of calories which the infant is getting per pound or per kilogram of body weight. To determine the energy quotient of the diet multiply the number of ounces of each food ingredient of the food mixture by their caloric values, add the products and divide the sum by the number of pounds or kilograms of the baby's weight.

CALORIC VALUES OF 1 OZ. (30 GM.) OF VARIOUS FOODS.

	Calories
Cow's milk	21
Human milk	21
16 per cent. cream	54
Skim milk	11
Buttermilk	11
Buttermilk mixture	21
Albumin milk	12
Chymogen milk	21
Keller's malt soup	25
Cane-sugar (by weight)	120
Maltose-dextrin compounds (average)	110
Malt-soup extract, dry, by weight	90
" " " by measure	132
Flour, by weight	100
Cereal waters (1 oz. cereal to quart)	3

PRACTICAL APPLICATION OF MILK DILUTIONS WITH ADDI-
TION OF CARBOHYDRATES IN INFANT FEEDING.

In the application of the rules for the feeding of normal, healthy infants, it must be remembered that each infant must be fed to meet its individual requirements, and the rules modified so as to meet the demands of the individual baby. If milk dilutions, with the addition of carbohydrates are used, the simplest and most natural standard would be one that would tell us how much milk and car-

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NOTE:—

1 gram of fat	= 9.3 calories.
1 " " protein	= 4.1 "
1 " " carbohydrate	= 4.1 "

bohydrates per pound or per kilogram body weight the baby should get. *To be exact we should express, or at least be aware, of the number of grams of proteins, fats, carbohydrates and salts that the infant is receiving for each pound of its body weight. We believe that if statistics on infant feeding were collected on this basis rather than in percentages of the ingredients in the milk mixtures (the total mixture being of such variable quantity) the collected data would be far more valuable as a basis for future work in infant feeding.*

DATA AS TO FOODS AND FOOD REQUIREMENTS USED AS A BASIS FOR ESTIMATING THE DIET OF INFANTS.

Average cow's milk contains the following percentages:

Fat	4.0	per cent.
Protein	3.5	"
Carbohydrates	4.5	"
CaO	0.172	"

Grams of food elements needed as a *minimum* by the average artificially fed normal infant in twenty-four hours:

	Per Pound.	Per Kilo.
Fat	1.5 to 2.0	3.3 to 4.4
Protein	1.5	3.3
Carbohydrates	5.0	11.0
CaO	0.08	0.17
Water	90.0	200.0

The milk or cream and skim milk needed to supply fat and protein will average 2 grams of sugar so it will be necessary to add amount needed in excess of this $\frac{1}{10}$ ounce or (3 grams) per pound or 6.6 per kilo.

For each gram of food elements in the mixture the following ingredients must be added:

Fat	$\frac{2}{10}$ ounce or 6 mils of cream.
	$\frac{5}{6}$ ounce or 25 mils of milk.
Protein	1.0 ounce or 30 mils of milk or skim milk.
Carbohydrates	$\frac{1}{30}$ ounce or 1 Gm. of sugar.
CaO	18.5 ounces or 600 mils of milk or skim milk.

For each pound of body weight the following will be required:

Fat (1.5 to 2 Gm.) .... ⁹⁄₁₀ to ¹²⁄₁₀ ounce or 9 to 12 mils of cream.

1¼ to 1⅔ ounces or 37.5 to 50 mils of milk.

Protein (1.5 Gm.) .... 1½ ounces or 45 mils of milk or skim milk.

Carbohydrates (3 Gm.) .. ¹⁄₁₀ ounce or 3 Gm. of sugar.

CaO (0.08 Gm.) .... 1½ oui ·es or 45 mils of milk or skim milk.

For each kilogram of body weight the following will be required:

Fat (3.3 to 4.4 Gm.) ..... 20 to 27 mils of cream.

85 to 110 mils of milk (average 100 mils).

Protein (3.3 Gm.) .... 95 mils (milk or skim milk).

100 mils in round figures.

Carbohydrates (6.6 Gm.).. 6.6 Gm. of sugar.

CaO (0.172 Gm.) .... 100 mils in round figures.

No allowance has been made for protein in cream.

Protein figured at 3.5 per cent. in milk.

Therefore to meet *protein* and *fat requirements* in feeding with diluted whole milk, the average normal infant will require each day a *minimum* of 1½ ounces (45 mils) per pound or 100 mils per kilo of body weight, exclusive of the sugar and starch which are added in preparation of the mixture.

Practical experience has taught us that infants under five months of age will frequently require amounts approximating 2 ounces (60 mils) of cow's milk per pound body weight, except during the first few weeks of life, when smaller quantities of whole or skim milk are indicated (see page 151). With the institution of a mixed diet, the infant thrives with less milk per pound body weight.

In beginning feeding with cow's milk, mixtures must always be started as weak formulæ, more often using only 1 ounce (30 mils) of cow's milk to a pound body

weight, gradually increasing the strength to meet the infant's needs.

Underweight infants should at first be fed according to their present weight, gradually increasing the strength of the mixture as rapidly as consistent with the baby's ability to handle the diet, and thus approximating the needs of a full weight baby of the same age. These babies will frequently take over 2 ounces (60 mils) of milk per pound body weight.

Number of Feedings in Twenty-four Hours. Three-hour intervals at the start, with 7 feedings in twenty-four hours, for the first month (6-9-12-3-6-10-2), 6 feedings during the second and the third month (6-9-12-3-6-10), 5 feedings by the fourth to the fifth months (6-10-2-6-10), according to the individual needs of the child.

Premature and delicate infants with a tendency to vomit are exceptions, and may be fed smaller amounts at more frequent intervals, even two hours, if indicated. Catheter feeding may be necessary, in which case the longer interval will usually answer.

Amounts at Each Feeding. On 3-hour intervals the quantity should be *one ounce more than baby is months old.* Example: A 3-months-old baby takes four ounces per feeding if fed every three hours.

On four-hour intervals, the quantity is *two ounces more than the baby is months old.* Example: A 6-months-old baby takes eight ounces per feeding on a four-hour schedule.

The increases per feeding are made gradually until the feedings reach eight ounces.

Exceptionally, infants cannot take this amount at each feeding, and when vomiting is the result of overfeeding, the quantity can be reduced and an extra meal substituted. Some infants will demand larger feedings.

After the fourth month the average infant will take daily 1 quart of the food mixture.

By the sixth month four meals of 8 ounces each of milk mixture may be given, and a fifth meal of broth and vegetables (*see rules for mixed diet, page 145*).

Water to be Added. In our own experience we have found that a concentrated milk mixture does not disturb the infant's digestion when the milk is boiled or alkalinized by sodium citrate, sodium bicarbonate, or lime-water. The amount of water is calculated by multiplying the number of feedings by the amount of each feeding, and subtracting the milk to be given.

Example: Baby aged 3 months should receive 5 feedings, 5 ounces each (age in months plus 2) or a total of 25 ounces for the day. Subtracting 16.5 ounces (11 pounds body weight and 1.5 ounces of milk for each pound) gives us 8.5 ounces as amount of water to be added.

The total fluids for the day should equal at least one-sixth the body weight. If the mixture does not total this amount sufficient water can be fed between meals.

Carbohydrates to be added. Having the necessary amount of milk and water, we ascertain the carbohydrate to be added.

Cane sugar answers our requirement for most cases.

Milk sugar acts as a laxative in many infants. Unless the laxative effect is desirable, it has no advantages.

Maltose and dextrin compounds are acceptable to the infant's digestion in relatively larger quantities. They are not as sweet as cane sugar. They are of practical value when large amounts of cane or milk are not well taken. Because of their varying maltose, dextrin and alkali content some are constipating and others are laxative. This must be given due consideration in their selection (see page 131, also see Appendix).

Cane and milk sugar are added in such quantities that the normal infant in its food mixture receives a total of from 4 to 6 grams of carbohydrates per pound of body weight per day, including that contained in the milk in the mixture. As one and one-half ounces of milk con-

tains approximately 2 grams of lactose it will be necessary to add from 2 to 4 grams of carbohydrates for each pound of body weight to the diet besides that contained in the milk. The needs of the average normal infant in the first months are usually satisfied by the addition of one-tenth of an ounce (3 grams) of cane sugar per pound body weight to the milk and the water in the mixture.

When using maltose-dextrin compounds somewhat larger quantities can often be fed to advantage. One-eighth ounce (4 grams) per pound are usually well taken.

We do not hesitate to add cereal water to the diet after the infant is one month old, and find it especially valuable in those cases in which we are feeding cane-sugar, and in which the infant takes a dislike to its food because of the intense sweetness of the mixture. One-sixtieth to one-thirtieth of an ounce (0.5 to 1.0 gram) of starch for each pound of body weight may be added to the mixture. This is best given in the form of cereal waters or well cooked cereals.

In underweight infants the amount of sugar to start with should be calculated on the basis of the present weight, approximating the quantity needed for a full weight infant as rapidly as the sugar tolerance permits.

The following table gives equivalents of 1 ounce by weight and the domestic measures of carbohydrates used in artificial feeding of infants:

	By weight			By measure	Table-spoonfuls	Dessert-spoonfuls	Tea-spoonfuls
					leveled	with a	knife.
Cane-sugar	1	oz.	30 Gms.	1.00 oz.	2	3	6
Milk-sugar	1	" "	"	1.50 "	3	4.5	9
Maltose-dextrin .	1	" "	"	1.50 "	3	4.5	9
Flour (wheat) ..	1	" "	"	2.25 "	5	7.5	15
Flour (barley) .	1	" "	"	1.50 "	3	4.5	9
Barley (pearl) .	1	" "	"	2.50 "	5	8	15
Oats (rolled) ..	1	" "	"	2.50 "	5	8	15

1 tablespoonful = 1.5 dessertspoonfuls = 3 teaspoonfuls.

To Break the Curd to Assist Digestion of Cow's Milk. Many infants can digest raw cow's milk. When not well taken, the tendency to formation of large protein curds is relieved by boiling the milk[1] from two to three minutes over the flame, or, better, by putting in a double boiler and heating until the water in the outer vessel boils eight minutes. Although the curd is less finely divided by the use of the double boiler, as compared with boiling on the direct flame, it answers the purpose of most infants, and causes fewer changes in the milk.

Addition of sodium citrate to the milk mixtures also prevents formation of hard protein curds. Bosworth and Van Slyke have shown that increasing amounts of sodium citrate added to the milk increases the coagulation time up to the point when 1.7 grains (0.1 Gm.) per ounce (30 mils) is added, after which the milk does not coagulate at all. Sodium which is added replaces some of the calcium in the caseinate, and forms calcium-sodium caseinate, and when rennin is added this double salt is changed to calcium-sodium-paracaseinate, which in the presence of sufficient quantity of sodium does not curdle. Sodium citrate may be prescribed either in 5-grain tablets, adding approximately 1 grain for each ounce of milk in the mixture, or a prescription may be written in such form that each teaspoonful will contain sufficient sodium citrate for the day's food.

When lime-water is added to cow's milk until it is neutral or faintly alkaline to phenolphthalein, a basic calcium casein is formed which is not acted upon by rennet, and will not form a curd, even in the presence of lime salts (Van Slyke). Casein is not coagulated by rennin when the solution is alkaline. When a sufficient amount of an alkali is given, the milk mixture remains neutral or alkaline in the stomach, even after the stomach has secreted acid, and large protein curds do not form then.

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[1] Brenneman, J.: Boiled versus Raw Milk, J. A. M. A., lx, 575. 1913.

Lime-water is commonly used in amounts equaling 5 per cent. of the milk in the mixture (1 ounce to 20 ounces of milk).

Not infrequently we have found the adding of citrate of soda or lime-water to boiled milk of advantage in the difficult feeding cases, and in the presence of vomiting.

Mixed Diet for Young Infants.

As early as the second or third month, 1 or 2 teaspoonfuls of orange juice may be given daily. This in part at least counteracts the effect of boiling. Start with one-half teaspoonful diluted with water, twice daily, and increase gradually until the juice of a whole orange is given.

Fifth month, a little well cooked cereal may be added to one of the meals (begin with 1 teaspoonful), adding part of the bottle of milk to it, the meal being finished by the remainder of the bottle, or it may be given in the bottle. Increase until 1 to 4 tablespoonfuls are added, once or twice daily.

At sixth month, infants readily take a broth and vegetable meal as a substitute for one of the milk feedings, in the form of a vegetable and meat soup. Begin with 1 ounce, and follow by a second bottle containing the milk mixture with 1 ounce less than full feeding. Gradually replace an entire milk feeding.

Ninth month, a vegetable soup or a clear broth (chicken, lamb, or veal), and toast or zwieback crumbs, with an additional portion of stewed fruits (apples, prunes) or a strained vegetable (spinach, carrots, potatoes, asparagus tips, peas, celery, beets, or turnips). The broth is usually given in the same quantity as the bottle, if given alone, or somewhat less if either the tablespoon of vegetable or fruit is given in addition.

Spinach, carrots or other vegetables may appear in the stool apparently unchanged, unless they are finely puréed. This may be disregarded if there are no signs of intes-

tinal irritation as the salts and vitamines are usually extracted, the colored cellular part remaining. It is to be remembered that the tubers, such as potato, are in reality largely starch and although valuable for their mineral content, they do not replace root or leaf vegetables in the diet.

CALORIC VALUES OF FOODS.

	Amount	Cal.
Apple sauce	1 ounce	30
Bacon (slice)	⅛ ounce	30
Bread	average slice, 33 Gm.	80
Butter	1 pat (⅓ ounce)	80
Cereal (cooked)	1 heaping tablespoonful (1 ounce)	50
Carrots (cooked)	1 ounce	13
Crackers (soda or Graham)	1 ounce	100
Cream (16 per cent.)	1 ounce	54
Custard	1 ounce	60
Egg	1 (1.5 ounces)	80
Egg (white)	1	30
Egg (yolk)	1	50
Gelatin	1 ounce	50
Malt extract	1 ounce	89
Meat	1 ounce	50 to 70
Milk (whole)	1 pint	350
Milk (whole)	1 ounce	21
Potato (whole)	1 medium sized	90
Potato (mashed)	1 heaping tablespoonful	70
Rice (boiled)	1 tablespoonful	60
Soup (vegetable)	1 ounce	15
Soup (chicken)	1 ounce	8
Toast	average slice	80
Vegetables (peas, beans, carrots)	1 heaping tablespoonful	30
Vegetable (cooked spinach)	1 heaping tablespoonful	16

These caloric values are approximate for the most part, but are sufficiently accurate for practical purposes. Thus the caloric value of a particular menu is easily figured.

Feeding Example No. 1. *Normal Infant, Age Three Months.* The infant should weigh 11 pounds (average birth-weight 7 pounds, plus 4 pounds, representing a gain of 5 ounces weekly for thirteen weeks).

Estimating 1½ ounces of milk per pound body weight, give 16½ ounces of milk.

Adding *three grams of cane sugar per pound* or 1 ounce for each ten pounds, is 1.1 ounces of sugar, or 2¼ level tablespoonfuls per 11 pounds

The infant should be fed five or six times daily and should receive at each feeding 5 ounces of mixture (age in months, plus 2 ounces). To make the total daily quantity 25 or 30 ounces as desired it is necessary to add 8½ or 13½ ounces of water to the quantity of milk as given above.

For practical purposes the cow's milk may be considered as averaging:

Proteins	3.5 per cent.
Fat	4.0 " "
Carbohydrates	4.0 " "

We will now calculate the amounts of the various elements in the mixture and the grams of each and calories per pound body weight in the milk mixture as given above for a 3 months old infant, weighing 11 pounds.

	Protein	Fat	Carbo-hydrate	Salts	Cal.
Milk, 16.5 ozs.= 495 mils ..	17.3	19.8	19.8	3.46 Gm.	346
Water, 8.5 " =255 " ..	....	....	....	.... "	...
Sugar, 1.1 " = 33 Gm. ..	....	....	33.0	.... "	132
Total mixture, 25 ozs.=750 mils ..	17.3	19.8	52.8	3.46 Gm.	478
For each pound body weight .	1.575	1.8	4.8	0.31 "	43

We thus find that the infant fed on the prescribed diet receives 25 or 30 ounces of the mixture containing:

Protein 1.575	Gm. per pound body weight
Fat 1.8	" " " " "
Sugar 4.8	" " " " "

The mixture containing the following percentages if made up for 25 ounces, (It will be noted that the percentages of the food elements per ounce of the mixture vary directly with the amount of water added and is therefore an unreliable basis for calculation.) and 43 calories per pound of body weight, all of which may be considered as a safe minimum.

Protein .... 2.3 per cent.
Fat .... 2.64 "   "
Sugar .... 7.00 "   "

A fruit juice should be included in the diet (page 145).

It may be repeated that the needs of the Individual Infant are to be covered by the Diet, and food of Higher Caloric Value per pound body weight is needed by some Infants. (Pages 140, 141 and 150.)

The mixture may readily be strengthened to meet indications for more fat and protein by the addition of milk, and more carbohydrate by the addition of flour and sugar.

Form for Directions for Preparation and Feeding.

This feeding formula may be given to the mother in the following form with directions for its further preparation attached.

Name............................   Age..........
        Milk .... 16.5 ounces
        Water .... 8.5 ounces
        Cane sugar .... 2.25 level tablespoonfuls
        (Milk Sugar: 3⅓ level tablespoonfuls.)

Place the above mixture in a double boiler, and starting with the water and the milk mixture cold, allow it to remain on the stove until the water in the outer vessel boils for 8 minutes. A small double boiler is preferable so as to obtain a deep column of milk, thereby holding the milk at a simmer.

Feed 5 ounces at each feeding, five feedings at 4 hour intervals: 6 a.m., 10 a.m., 2 p.m., 6 p.m., and 10 p.m.

Not infrequently while the ingredients in the diet fully meet the infant's needs for growth and development, the amount of the individual feedings are insufficient to satisfy the appetite. The infant can usually be satisfied in such instances by adding more water or cereal water to the mixture, thereby increasing the quantity of the individual bottle without materially altering its quality.

It should also be remembered that some infants may require more milk than 1½ ounces per pound body weight, or again, occasionally an infant cannot take a formula as strong as that given above, because of an inability to handle one or more of the cow's milk elements, more commonly fat, protein or the excess of salts. In all exceptional cases the individual infant must necessarily have its needs fulfilled, by an increase or decrease in the milk components.

Feeding Example No. 2. *Normal Infant, Age Eight Months.* The infant should weigh 17.25 pounds (average birth-weight 7 pounds), which should be doubled in the first five months (14 pounds), plus a gain of 4 ounces a week for the remaining thirteen weeks (3.25 pounds).

The following mixture will be prepared:

1.5 ounces of milk per pound body weight, equals 26 ounces.

Water to make one quart, equals 6 ounces.

Sugar 1.5 ounces. As stated on page 130 the amount of sugar to be added is usually limited to 1.5 ounces; further carbohydrate needs being furnished by the addition of cereal waters or cereals.

Starch 0.25 ounce or 8 grams (approximately $\frac{1}{60}$ ounce or 0.5 grams per pound).

This to be fed in four feedings of 8 ounces each, and the fifth may be replaced by a soup and vegetable meal. A small cereal feeding (2 to 4 tablespoonfuls) can also be given with 1 or 2 of the bottles, pouring part of the bottle of milk over it, and finishing the meal on the remainder of the bottle. (Mixed Diet, page 145.)

	Protein	Fat	Carbo-hydrate	Salts		Cal.
Milk, 26.0 ozs. = 780 mils ..	27.3	31.2	31.2	5.46	Gm.	546
Water, 6.0 " = 180 "	.. ....	....	....	....	"	...
Sugar, 1.5 " = 45 Gm. ..	....	....	45.0	....	"	180
Starch, 0.25 " = 8 "	.. ....	....	8.0	....	"	25
Vegetable soup, 8.0 " = 240 mils ..	2.0	4.5	8.0	2.4	"	144
Cereal, one heaping tablespoon-ful, 1.0 " = 30 Gm. ..	....	....	15.0	....	"	50
Total feeding	29.3	35.7	107.2	7.86	"	945
For each pound body weight .	1.7	2.1	6.2	0.46	"	55

Further needs of the individual case can be supplied by concentrating the milk until 1 quart whole milk is given, the carbohydrates in the mixture being gradually decreased and given in another form, as gruel, custard, etc.

Feeding Example No. 3. *Underweight Infant, Age 3 months. Weighing 8 pounds.* To start with, prepare the following mixture:

Milk 12 ounces. (1½ ounces per pound present weight).

Water 13 or 18 ounces. Sufficient to make 5 or 6 feedings of 5 ounces each.

Cane sugar 0.8 ounces (1½ level tablespoonfuls) (⅒ ounce or 3 grams for each pound).

To meet the requirements of this infant for growth and development, the needs of a full weight infant of the same age must be approximated as rapidly as the infant's tolerance for food permits. These increases can usually be made rapidly, if the infant is well other than for its underfeeding. The first increases being made in the carbohydrates in the form of further addition of sugar and cereal waters, until 3 grams per pound of sugar and 0.5 to 1.0 gram per pound of cereal flour in the form of cereal water are added, calculated on the basis of average full weight, 11 pounds, for this age.

The milk can be increased until 1½ ounces per pound of full weight, or 16.5 ounces for the total mixture are given.

If the infant is suffering from digestive disturbances, it may be necessary to start with 1 ounce of milk or even less per pound of its present weight, that is 8 ounces or less in the mixture, adding only 1 or 2 grams of sugar per pound. It must, however, be remembered that the infant will require 32 calories per pound body weight to sustain it. And if it is underfed for too long a period, it will result in inanition.

Artificial Feeding During the First Weeks of Life. The rules as given for infant feeding are hardly applicable for feeding during the first one or two to three weeks of the infant's life. The infant's first feedings should consist of higher dilutions of either whole or skim milk, should be boiled, and sugar added in smaller percentages than suggested for the older infants. Such mixtures must of necessity show a lower caloric value than will meet the infant's needs for growth and development, but, as suggested, the mixture for the newborn should be composed of weak formulæ, and increased according to the infant's tolerance. The following table of mixture will act as an outline for average cases:

DIET FOR NEWBORN INFANTS DURING THE FIRST FOUR WEEKS OF LIFE.

	1st 48 hours	3-4 days	5-6 days	7-8-9 days	10-11-12 days	13-14 days	3d week	4th week
Milk (whole), ozs.	..	..	...	3	4	6	8	11
Milk (skim), ozs.	..	6	8	5	4	4	2	...
Sugar (cane), dr.	1	1	2	2	2	3	4	6
Water (boiled), ozs.	16	10	8	8	8	8	8	10
Calories in mixture	15	81	118	148	158	215	250	321

Feedings:

Amount in ozs.	1	2	2.5	2.5	2.5	3	3	3.5
Number daily	6	6	6	6	6	6	6	6
Intervals in hours	4	4	4	4	4	4	4	4

The mixtures previously mentioned should be boiled for three minutes over the direct flame or in a double boiler. If the latter is used, the water in the outer vessel should be boiling for eight minutes. Add boiled water to make up the original quantity.

Method of Feeding a Baby from the Bottle. Babies should be fed while they are lying on their beds, the upper part of the body being somewhat elevated by means of a pillow of proper thickness. The baby should be turned slightly on the right side, as it has been found that the stomach empties itself sooner in that position.

The bottle should always be held by the nurse or attendant, until it is empty. From fifteen to twenty minutes should be occupied with the meal.

Do the above rules furnish mixtures of a quality and quantity proper to meet the infant's needs? If proper mixtures they should

   (1) Contain approximately

Protein .... 1.5 to 2.0 Gm. for each pound of body weight
Fat .... 1.5 " 2.0 " " " " " " "
Carbohydrates .. 4.0 " 6.0 " " " " " " "

   (2) Calories per pound body weight for normal
        infant:

        Under 2 months of age ...... 30 to 45 calories
        Over 2 months of age .... 45 " 55 "

   (3) Percentages in the mixtures.

It is well to know the percentages of the various ingredients in the diet, as they will assist in the proper interpretation as to the etiology of food disturbances.

Fat. Infants, according to their age, under normal conditions, digest from 2 to 3.5 per cent. of fat. Some infants digest fat badly, consequently in some cases it is necessary to give skim milk.

Proteins. In the average feeding mixture for infants under 10 months, 2 to 3 per cent, of proteins are well taken.

Carbohydrates. They should, as a rule, not exceed 6 to 7 per cent., the average amount in human milk, including the sugar contained in the milk before its modification.

Summary.

I. Preparation of the mixture.

1. Calculate the baby's normal weight.

2. Calculate the amount of cow's milk to be used in the preparation of the mixture, taking 1.5 ounces of cow's milk per pound of normal body weight at that age, which is a safe minimum for a healthy infant.

3. Calculate the total daily amount of the mixture by multiplying the amount of each feeding (age in months plus 1 or 2 ounces) by the number of feedings.

4. Add water to make the mixture up to this total amount.

5. Add 3 grams of sugar, and later 0.05 to 1.0 gram of starch for each pound of body weight.

6. Make the curd more digestible by boiling or alkalinizing the mixture or using cereal water as a diluent.

II. Checks on the above mixture.

1. Number of grams per pound body weight of each food ingredient in the mixture.

2. Total caloric value of mixture and caloric value per pound body weight.

III. Remember that—

1. Orange juice or codliver oil additions to the diet should be started by the second or the third month.

2. When more than 1 quart of milk mixture is needed to properly nourish the infant, most infants have reached the age when a mixed diet should be instituted.

3. These amounts are relative, and must be increased or decreased according to the infant's progress and individual needs, *the above rules furnishing a safe minimum for a healthy infant.*

4. The above amounts are usually insufficient for the underfed infant after it has become accustomed to the diet. Frequently it is necessary to approximate the requirements of a normal baby of that age.

5. Premature and underfed infants must at first be fed smaller amounts.

6. *The food formula of a baby clinically healthy and making a satisfactory gain in weight should not be changed without a well-defined indication.*

EXPLANATORY NOTE. For practical purposes we have used pounds for weight, and ounces for measuring fluids, because of the common use in the home of avoirdupois scales, and bottle and measuring glass graduated in ounces. We have also calculated 1 oz. = 30 Gm., and 2.2 lbs. = 1 Kg.

CHAPTER V.

CREAM AND SKIMMED MILK MIXTURES.

By the use of 16 per cent. cream and skimmed milk as the basis for obtaining various milk modifications, a wide range of combinations of the various food elements can be obtained.

For feeding purposes, *gravity cream,* of which about 6 ounces or somewhat less may be obtained from a good quality of milk contains fat 16, protein 3.5 and carbohydrate 4.5 per cent. The *skimmed milk* may be obtained by carefully pouring off or dipping the cream. If this is carefully performed, it should contain F 0, P 3.5 and C 4.5. Skimmed milk obtained by this method is not entirely fat free. However, any cream remaining in the lower portion would be balanced by a lesser quantity of fat in the cream used and the end result in making these mixtures would be about the same.

The average infant should receive fat 1.5 to 2 Gm., protein 1.5 Gm. and, as a *minimum* of added carbohydrate 3 Gm. (above that contained in the cream and skimmed milk) per pound body weight. These will be obtained by the use of cream (16 per cent.), skimmed milk and sugar, which contain the following:

Cream (16 per cent. fat), in 1 ounce, 5 grams of fat.

Skimmed milk (3.5 per cent. protein), in 1 ounce, 1 gram of protein.

Sugar (100 per cent. carbohydrate), in 1 ounce, 30 grams of carbohydrate.

The following amounts will be needed:

For each gram of fat, add $\frac{1}{5}$ ounce, or 6 mils of cream.

For each gram of protein, add 1 ounce, or 30 mils of skimmed milk.

For each gram of carbohydrate, add $\frac{1}{30}$ ounce, or 1 gram sugar.

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In the mixture the ingredents will be used in the following amounts *per pound body weight:*

Cream, 3/10 to 4/10 of an ounce (Fat, 1.5 to 2 grams).
Skimmed milk, 1½ ounce (Protein, 1.5 grams).
Sugar, 1/10 of an ounce (Carbohydrates, 3 grams).

In the mixture the ingredients will be used in the following amounts *per kilogram body weight:*

Cream, 27 mils (Fat, 4.4 grams).
Skimmed milk, 100 mils (Protein, 3.3 grams).
Sugar, 6.6 grams (Carbohydrates, 6.6 grams).

In the underweight infants the amounts would be calculated on the basis of present weight, at the beginning, but these would be increased gradually to the amounts necessary for a normal weight infant of the same age.

Example: Given a ten pound baby, and desiring to feed the required amounts of fat, protein and carbohydrates, which may be taken as 20 grams of fat, 15 grams of protein, and 30 grams of carbohydrate for one day's food, these quantities would be supplied by:

Cream .... 4 ounces.
Skimmed milk 15   "
Sugar 1   "
Water 8   "

The small excess of protein in the cream may be considered as a negligible quantity.

It is readily seen that by thinking of the food requirements per pound or kilogram body weight of the infant that the danger of over or under feeding is minimized as compared with the method so commonly in use by which the food ingredients are calculated on the basis of the percentage content in the mixture.

There can be no doubt as to the accuracy of the modifications which can be obtained by this method of feeding, but as previously suggested, it has the disadvantage of requiring considerable calculation due to the use of cream and skim milk in the place of whole milk and in actual experience the disadvantages to healthy infants of the

possible excess of protein in mixtures made with simple dilutions of whole milk have been exaggerated. Practical experience presents convincing evidence that far more infants develop gastro-intestinal disturbances from feeding excessively rich cream mixtures. The greatest objection to high milk feeding is high protein constipated stools which can be overcome by adding more sugar.

CHAPTER VI.

FEEDING IN LATE INFANCY AND EARLY CHILDHOOD.

Mixed Diet After the Fourth Month. (See page 145.)

Feeding During the Last Quarter of the First Year. The following diet list will serve as an example for feeding during this period:

Nine to twelve months diet.

6.00 A.M.   Milk mixture, 8 ounces. Milk, 6 ounces; water, 2 ounces; sugar, 2 level teaspoonfuls.

8.30 A.M.   Orange or prune juice, ½ to 1 tablespoonful (0.25 to 0.5 oz.). If preferable, this may be given with the 10 A.M. or 2 P.M. meal.

10.00 A.M.   Milk mixture, 8 ounces. Cereal (farina, oatmeal, etc.), 2 to 4 tablespoonfuls.

2.00 P.M.   Vegetable soup or a clear broth (chicken, lamb or veal), with an additional portion of a strained vegetable (spinach, carrots, potatoes, etc.). Vegetables can be started by the ninth month. The broth is usually given in the same quantity as the bottle, if given alone, or somewhat less if a vegetable is given in addition. When starting the soup feeding, first replace 1 ounce of the 2 P.M. bottle by 1 ounce of soup in another bottle; then give 7 ounces of the milk mixture. Gradually increase soup and diminish milk until an entire bottle of milk is replaced by soup. Gradually cut water and sugar out of the milk mixture until full milk is given by the tenth or eleventh month.

6.00 P.M.   Milk mixture, 8 ounces, and bread, zwieback crumbs or cereal.

10.00 P.M.   Milk mixture, 8 ounces, if needed.

A slice of crisp bacon may be given to advantage during the eleventh and the twelfth months, probably best with the mid-morning meal.

NOTE: For preparation of diets see Appendix.

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Four feedings a day are usually sufficient during the early part of the second year.   In such a diet the fruit juices which may be given once or twice a day should not be considered as meals, and may be given between the regular feedings.   Whole milk is now fed, and should not exceed 1 quart daily.   The sugar and water are decreased gradually.

Twelve to fourteen months diet.

6.00 A.M.   Milk, 8 ounces.

8.30 A.M.   Orange juice, prune juice, or apple sauce (1 oz.) If preferred, this may be given with the 10 A.M. or 2 P.M. meal.

10.00 A.M.   Milk, 8 ounces, and cereal (farina, oatmeal, etc.) 4 or 6 tablespoonfuls, slice of crisp bacon.

2.00 P.M.   Vegetable or cream soup and zwieback, toast, etc., or a clear broth (chicken, lamb or veal), with additional portion of 2 to 4 tablespoonfuls of strained vegetable (spinach, carrots, potatoes, etc.).   The broth is usually given in the same quantity as the bottle, if given alone, but somewhat less if a vegetable is given in addition.   A little scraped beef or beef juice may occasionally be added to the vegetable.   Most infants may be given small amounts of coddled egg.

6.00 P.M.   Milk, 8 ounces, and bread, zwieback or cereal, custard or pap.

10.00 P.M.   Milk, 8 ounces, if needed.

No milk should be given with the noon meal.

After the twelfth month cow's milk should be limited to from 1½ to 2 pints daily.

Fourteen to eighteen months diet.

6.00 A.M.   Milk 8 to 10 ounces

8.30 A.M.   Fruit juice (orange juice, prune juice, or apple sauce) 1 to 2 ounces.

10.00 A.M.   Cereal, 4 to 6 tablespoonfuls, with 2 ounces of milk or cream, followed by 6 to 8 ounces of milk.   Toast, zwieback, crackers, or wafers may be alternated with bacon.

2.00 P.M. (1) Vegetable or cream soup and zwieback or toast, or (2) a clear broth (chicken, lamb or veal), with an additional portion of four table-spoonfuls of vegetable mixture (spinach, carrots, potatoes, etc.). The broth is usually given in the same quantity as the bottle, if given alone, but somewhat less if the vegetable is given in addition.

Part or whole of a coddled egg with toast, zwieback or cracker crumbs can now be added to the above soup and vegetable meal.

The egg may be alternated with beef juice or scraped beef.

6.00 P.M. Cereal, 4 tablespoonfuls, farina, cream of wheat, oatmeal, arrowroot, custard or pap, with 8 ounces of milk. Part of the milk may be given over the cereal, or as bread and milk, or milk toast. Jelly or honey with bread.

10.00 P.M. Milk, 8 to 10 ounces. (Can usually be left out by this time.)

Eighteen months to three years.

7.00 A.M. ·Stewed fruit or orange juice; cereal; crisp bacon, alternate with soft boiled or poached egg; Bread and butter or toast; milk or weak cocoa.

12 or 1 P.M. (1) Broth: meat or vegetable soup thickened with cereal. (2) Meat: lamb chops, scraped beef, chicken or beef juice. (3) Vegetable: baked or mashed potatoes; strained spinach, carrots, turnips or celery. (4) Dessert: gela-tine, custard, cornstarch or rice-pudding, or other simple dessert.

6.00 P.M. Cereal and bread or cracker with milk. Baked apple, apple sauce or other stewed fruit.

Other Foods Permitted at Three Years.

Meats. Broiled or boiled fish, roast or stewed poultry, raw or stewed oysters, broiled beefsteak, roast or broiled beef or mutton—all in moderate quantities.

Eggs. Soft boiled, poached or scrambled, 1 or 2 daily.

Cereals and Breads.   Oatmeal, hominy grits, wheaten grits, cornmeal, barley, rice, macaroni, etc.   Light and not too fresh wheat and graham bread, toast, zwieback, plain unsweetened biscuit.

Soups.   Plain soup and broth of nearly every kind, preferably vegetable broth.

Vegetables.   White potatoes, boiled onions, spinach, carrots, peas, asparagus (except the hard part), stewed celery, young beets, arrowroot, tapioca, sago.

Fruits.   Nearly all, if stewed and sweetened.   Of raw fruits, peaches are the best; pears, grapes freed from seeds, oranges.

Desserts.   Light puddings, as rice pudding without raisins, bread pudding, plain custard, pap, wine jelly, ice cream, junket.

Foods to be Taken with Considerable Caution.
Muffins, hot rolls, sweet potatoes, baked beans, turnips, parsnips, cabbage, egg plant, stewed tomatoes, fresh corn, cherries, plums, raw apples, huckleberries, gooseberries, currants, preserved fruits.

Foods to be Avoided.   Fried foods of any kind, griddle cakes, pork, sausage, highly seasoned food, pastry; all heavy, doughy, or very sweet puddings; unripe, sour, or wilted fruit; bananas, cucumbers, nuts, coffee, alcoholic beverages.

PART IV.

Nutritional Disturbances in Artificially Fed Infants.

CHAPTER I.

MINOR DISTURBANCES.

1. Vomiting.

VOMITING is one of the most frequent symptoms in the nutritional disturbances of infancy and childhood, and will be reviewed from the standpoint of its association with this class of diseases, without reference to its occurrence in other systemic diseases.

Overfilling of the stomach is one of the most frequent causes of vomiting in infancy. The young infant vomits easily, and without effort. The weak sphincter at the cardia predisposes to regurgitation. Regurgitation of only a small portion of the meal is designated as "spitting." This latter symptom has become less common since the introduction of the longer feeding intervals, which allows the stomach to empty itself thoroughly before the next feeding. Other than too frequent feedings, too large an individual meal, and food too rapidly taken, are the most common causes of vomiting. These conditions are easily remedied.

Not infrequently overfilling of the stomach is due to swallowing of air during nursing. Some infants are especially prone to swallow large amounts at each feeding, and unless this is relieved, it will lead to vomiting. This condition is frequently seen in young infants who are left unattended to feed from a bottle. This results in their nursing air from the partially filled

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nipple as the bottle becomes empty. It should be an invariable rule to support the bottle in a semi-upright position for all infants until they are of such age as to enable them to properly hold the bottle for themselves. This will also insure their getting their food while still warm.

Placing an infant in an upright position, preferably with its chest against the mother's shoulder at the end of the nursing and avoiding at the same time compression of the abdomen, so that the air bubble may rise to the cardia and be expelled, offers the best solution for temporary relief. Some infants develop an extreme habit of air swallowing, and in these it may become necessary to put the infant in the erect position two or three times during the course of a nursing.

Excessive handling and abdominal bands that are too tight are frequently causes of vomiting.

Improper Diet. Excessive feeding with fat, such as is frequently seen in formulæ made from cream mixtures and top milk mixtures, are common causes of vomiting, and should lead to reduction of the fat contents in part by whole or skim milk. Excessive quantities of sugar in the diet may also cause vomiting. Vomiting due to the large tough protein curd of raw milk can be obviated by boiling or alkalinizing the milk.

Nervous vomiting or habit-vomiting is an exceedingly intractable and common form seen in many infants. In such cases the slightest excitement may produce vomiting; such as crying, sudden movement of the infant by the mother or nurse.

Spasm of the cardia or pylorus are frequent causes of vomiting more especially in early infancy. Spasm of the cardia is rarely diagnosed except by the radiograph. In our experience these cases are found more frequently than formerly supposed. These types of vomiting may appear immediately after birth, but usually do not develop for some days or even weeks, and sometimes not until the infant is several months old. In

the milder cases this may be the only symptom and the infants may obtain sufficient food, and thereby avoid any interference with proper nutrition. In both types the vomiting may be explosive. This is more common in the pyloric type or when the two are associated. The vomitus shows, as a rule, little or no evidence of disturbance of digestion. In extreme types nutrition will suffer and the hunger stool is present. The presence of increased and visible peristalsis in the pyloric type may lead to the diagnosis of organic stenosis. They can only be differentiated by the aid of a fluoroscope and radiograms following a bismuth meal, together with a careful study of the history of its development and the effect upon the infant's development.

Congenital hypertrophic stenosis of the pylorus is a frequent form of obstruction. Most frequently it comes into evidence between the second and the fourth week after birth. There is nothing characteristic about the vomiting in the beginning. It soon becomes forcible and explosive. The gastric contents may be shot out of the mouth to a distance of several feet. The vomiting usually occurs soon after the taking of food, but may occur at any time, sometimes not until just before the next feeding. Two or even more feedings, are sometimes retained and expelled together. The vomiting may be accompanied by pain. Constipation quickly develops, because so little of the food passes through the pylorus into the intestine, that there is but little residue to be passed out of the bowels. The stools are small and composed mainly of mucus. Increased peristalsis with visible waves traveling from the left to the right and ending at the pylorus with increasing size until vomiting occurs, or to the point of exhaustion on the part of the stomach, is a constant finding. In a large percentage of the cases, a small tumor may be felt at the pylorus when the infant is quiet and the abdomen is not too much distended.

Loss of weight is a constant symptom. The skin becomes dry, the face pinched and the baby soon shows all the evidences of starvation.

Rumination consists in repeated regurgitations of small amounts of the food taken and occurs some time after ingestion. Some of this is promptly swallowed again, but a considerable amount may be lost from the mouth. It usually lacks the forcible character that pertains to true vomiting. In the mild type the rumination occurs only at long intervals, perhaps once or twice a day, and then is not so marked as to be accompanied by any definite loss of food. This condition usually responds readily to proper hygiene and feeding, and causes very little anxiety. In the severe types we find a nervous, emaciated, pale child, which cries rather easily and does not sleep well. The food which is given the child is well taken. Soon after the bottle is taken, the child begins to bring the food up.

The food which is brought into the mouth is gargled for a short time and then swallowed. This may happen again and again, the interval between the attacks becoming exceedingly short. Some of the infants stimulate the attacks mechanically by putting the hands into the mouth, others by rotating or protruding the lower jaw. Commonly the regurgitation is initiated by a rigid extension of the body, a throwing back of the head, arching of the chest and contraction of the abdomen. Usually following the regurgitation the infant makes chewing movements upon such food as is not expelled and it is again swallowed. I have recently had such a case under observation, which showed a marked dilatation of the esophagus and cardio- and pylorospasm.

Acute gastric indigestion may also be caused by the ingestion of foods to which the infant is not accustomed, or by the accidental swallowing of foreign bodies.

Rarer types of **obstruction of the gastro-intestinal tract** are occasionally seen, such as congenital or inflammatory obstruction due to fibrous bands. Of the former I have recently seen two cases in which the lower end of the esophagus was obstructed by a band of adhesions, which was relieved by operation. Congenital atresia of the small intestine may be mistaken for pyloric spasm or stenosis. Intussusception, volvulus, appendicitis, diverticulitis and the different types of peritonitis are all accompanied by vomiting. In all cases of vomiting of sudden onset of unknown cause, examination should be made for incarcerated umbilical and inguinal hernias.

Treatment. The treatment of vomiting varies with its cause.

Spasm of the cardia or pylorus in the artificially fed is often cured by a change to human milk. When human milk is not available, feeding with a concentrated diet as thick cereal paste,[1] will frequently relieve the condition. Small feedings at more frequent intervals are often better retained. Re-feeding after vomiting may be necessary to avoid starvation. Boiling and alkalinizing the milk mixture is to be recommended. In the absence of organic obstruction, medication with atropine sulphate, paregoric and calcium salts may relieve the tendency to spasm. In the severer cases daily or even more frequent lavage with a weak solution of bicarbonate of soda is valuable. It has been our experience that in some of the cases feeding by gavage resulted in the disappearance of the tendency to spasm in both the cardia and pylorus type. In some of our cases rectal instillation of normal saline or Ringer's solution at regular intervals for the relief of the anhydremia has seemingly resulted in a lessening of the spasm probably through reflex action.

Congenital Hypertrophic Stenosis. The first indication for treatment is feeding small quantities of human milk

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[1] Thick cereal gruel—see Appendix.

at short intervals. The further dietetic and additional treatment is the same as that recommended for pylorospasm. Surgical treatment, when indicated, should not be too long delayed, because of the danger of too prolonged starvation. The Rammstedt or its modification is the operation of choice.

Rumination. The feeding of concentrated diets as thick cereal paste which increase the difficulty of regurgitation may alone result in a cure. In other cases mechanical methods may be employed such as splinting and pinning the arms down so that they may not reach the throat. The use of a dove-tail bandage about the chin and fastening over the head, or a cap made with tapes which fasten under the chin, as suggested by Batchelor[1] or plugging of the nostrils, all have been used with success, but will be attended with a certain number of failures. Radiographic study should be made of the esophagus, cardia and pylorus in every case, and treatment directed toward the relief of any anatomical anomalies which may be present. In older infants the psychic condition must be considered and all irritation and excitement avoided. In the hospital such an infant should be placed in a room by itself or screened from its immediate surroundings.

2. Colic and Flatulence.

Infantile colic is due to distention and increased peristalsis either in the stomach or intestine, or both, resulting in spasmodic contractions.

The colic period of infancy is chiefly the first three months. After this time the peculiar susceptibility gradually diminishes. The so-called "idiopathic" form of colic is more commonly intestinal but not infrequently the stomach alone may be the seat of origin.

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[1] Batchelor, M. D. and R. P.: Am. J. Dis. of Children. Vol. 17, 1919. 43.

The constant solicitude of the nurses, because the baby has "gas on the stomach" is unwarranted. All bottle-fed babies have gas in the stomach. They swallow it with their meals in the form of air. If the baby is gently raised to a sitting posture the gas will usually "come up." This may be done in the middle of a feeding if the stomach seems unusually distended.

Some infants, more especially those of the neuropathic type, seem predisposed to attacks almost from birth. They are the fretful, sleepless type of infants and seem at all times eager to take food. They usually insist upon sucking their hands or some other object and are air swallowers. More frequently colic is due to increased intestinal peristalsis, often secondary to over-distention. Many cases of colic appear to have a nervous origin brought about reflexly as, for example, through chilling of the surface of the body.

Constipation is very frequently associated with colic and flatulence, disappearing with the institution of a proper diet. As well as being a cause, the coexistence of constipation in the presence of other etiological factors, tends to aggravate the colic. Repeated purgation for an existing constipation is a frequent source of pain in the intestines. More commonly the habitual colic, as seen in the young infant, may be taken as an evidence of gastric or intestinal indigestion, and may be due to one of several causes: (1) Too much milk at proper intervals, (2) too frequent feedings, and (3) mixture too rich in fat, or (4) excessive in carbohydrates. Regurgitation and vomiting are commonly associated, and not infrequently diarrhea results. By a careful study of the diet and observation of the stools the offending factor can in most instances be eliminated. Excessive flatulence can frequently be eliminated by reduction or change in the kind of sugar and cereal gruels. A reduction in all the elements of the food may be temporarily necessary in the presence of severe symptoms.

Colic is a very common symptom in the pre-menstrual period of the mother and usually is present until a free flow is established. The infant should be left at the breast.

Acute attacks of enteritis, peritonitis, appendicitis, intussusception and volvulus must be excluded in sudden acute attacks.

Treatment must be directed to the elimination of the cause.

In the nervous type of infants, which are frequently termed "colic babies," this condition may persist for several months and the treatment must be directed to palliative measures. A cure will result only with improved development and when the digestive organs attain their normal functions. This class of infants are easily spoiled and should be placed under the supervision of a capable nurse, if the mother has not the proper temperament to combat the child's disposition.

Not infrequently the crying due to underfeeding may be interpreted as colic. Reduction of the diet of these infants is a source of danger. If the stools are good, and there is no vomiting, and the baby is gaining in weight, one should be convinced that it is not the cry of habit before making changes in the diet.

In breast-fed infants attention to the health of the mother or wet-nurse, avoidance of excitement, regular exercise and regulation of the bowels are necessary. In both breast-fed and artificially fed infants prevention of constipation and over-feeding of the infant, more particularly with high fat and carbohydrate mixtures are to be avoided.

If the infant is doing well, notwithstanding the colic, it should not be removed from the breast, nor should radical changes in the method of feeding be instituted without definite indications.

In feeding with raw milk the stools should be examined for protein curds. If they are present, the milk

should be boiled or alkalinized. Not infrequently the condition will be relieved in the artificially fed by substituting whole or skimmed buttermilk mixtures for sweet milk.

The addition of powdered casein in amounts varying from 5 to 20 Gm. dissolved in the day's milk mixture will often relieve colic, in all probability due to lessening of intestinal peristalsis. A similar result may be attained by the administration of calcium lactate two or three times daily, in doses of 0.3 to 0.6 Gm. (5-10 Grs.).

The addition of 3 to 5 mils of liquid culture of active lactic acid bacilli or a corresponding amount of an active culture in dry form is valuable in the treatment of many cases, both in the breast and artificially fed. It should be continued for some time.

The administration of mild carminatives in the form of camomile or fennel tea, peppermint water or aromatic spirits of ammonia (0.06-0.20 mil) (1-3 minims), or compound spirits of ether (0.25-0.30, 4-5 minims), are often effective as palliative measures. With the abdominal distention the most efficient means of relieving the intestinal tract is by massage or enemata. For the latter normal saline, weak soap suds or glycerine in water may be used. A glycerine suppository will usually accomplish the same result.

In prolonged attacks dry or moist heat may be applied to the abdomen together with a hot-water bag at the feet. In the severe cases an opiate may be needed for temporary use. Camphorated tincture of opium, in doses varying from 0.06 to 0.65 c.c. (1-10 minims), is the safest form of administration.

3. Constipation.

By this term is indicated a condition in which the number of evacuations are less frequent. smaller in amount or firmer and drier than would be normal for

the given infant. Constipation must be regarded as a symptom. The term is a relative one and as usually applied relates more to the character than to the frequency of the stool in the normal infant. In the presence of underfeeding or starvation the total amount must be given consideration in order to properly interpret its significance. Under normal conditions the breast-fed infant under six months of age will average two to four movements daily. While the artificially fed infant, except while on the carbohydrate rich diet, will usually average only one or two movements.

In the Newborn. The complete absence of stools due to congenital malformations of the gastro-intestinal tract, such as atresia of the esophagus or intestine, or imperforate anus, should not be classed as cases of constipation, and the cause can usually be ascertained by careful study of the history, time of onset and character of the accompanying symptoms and a roentgenological examination. Somewhat later the question of insufficient food as a cause of minimal stools must be considered. This may be due to mechanical obstruction due to spasm of the cardia or pylorus or hypertrophic stenosis at these orifices. The food taken in these conditions may be sufficient, but it is later lost through vomiting. On the other hand, insufficient food in the breast-fed during the first days or insufficient intake or improper diet in the artificially fed, may result in the so-called starvation stools which are frequently termed constipation stools, but are not properly so classed. Frequently the constipation is due to sluggishness of the intestines because of improper stimulation or lack of response to the mechanical irritation of the food due to muscular weakness or dilatation of the intestinal tract. This is most frequently seen in the lower part of the large intestine.

In Infancy. During this age a large number of factors may cause constipation. Not infrequently several factors are involved in a given case. In a proper con-

sideration of the subject it should be borne in mind that the cause of the constipation, once the food has entered the intestines may lie in any one of the segments of the tract, and may be due to insufficient secretion or excessive absorption, an improper diet or an atony of the intestinal wall.

Mechanical. Among the more common types several have already been mentioned. The most frequent cause in this group is the large size of the colon, more especially the sigmoid flexure and the rectum. This condition may be due to or result in an atony of the intestinal and abdominal walls, or may produce kinks which result in temporary obstruction of the lumen. Obstruction may also be due to conditions outside of the intestine, such as fibrous bands or adhesions, which latter may be of pre- or post-natal origin.

Reflex or Spasmodic. Reflex or voluntary retention of the stools may be due to painful anal conditions, such as fissures, ulcers or spasm of sphincter. The latter condition is frequently due to the accumulation of large, hard, dry fecal masses in the lower rectum, the more common causes of which will be described under Dietetic Errors.

Atony of the Intestinal Wall. This is one of the most important causes of chronic constipation and while not infrequently due to congenital causes, more commonly it develops after birth. Hyposecretion of the thyroid gland, represented in its extreme form in cretinism, is always associated with constipation due to atony of the entire intestinal tract. A similar condition is also seen in athrepsia, rickets and secondary anemia. All of these are usually associated with a weakness of the abdominal wall, which is an important predisposing cause. Affections of the central, peripheral and sympathetic nervous systems are infrequent, but important causes. Among these are chronic hydrocephalus, intracranial hemorrhage, inflammatory conditions of the brain and

spinal cord. Acute and chronic febrile diseases not involving the gastro-intestinal tract are usually accompanied by constipation. Improper training and lack of exercise result in increasing weakness of the intestinal and abdominal walls. The same result may follow the excessive use of cathartics and sedative drugs.

Dietetic Errors. Insufficient intake of food and water or an improper balance of the diet are the most frequent causes of constipation. Associated with these and to a large extent dependent upon them is a hyposecretion of the intestines, liver and pancreas. On the other hand, as will be described later, an excessive intestinal secretion may result from improper feeding and may in turn result in constipation, due to the formation of so-called soap stools. Food, especially its carbohydrate contents, is the normal stimulant of the intestine.

In the breast-fed infant receiving a sufficient quantity of milk we rarely see true constipation because of its relatively high sugar content. The condition described as constipation in the breast-fed infant is usually due to disproportion between the peristalsis in the lower bowel and over-action on the part of the anal sphincter. In the underfed breast-fed infant, however, true constipation may develop, due to insufficient stimulation of the intestines. In the latter class of cases, however, not infrequently numerous stools are passed which consist mainly of dark colored mucus. This represents the normal secretion of the colon mixed with the color of biliary pigments and a minimum of food residue. Similar stools may be passed by the underfed infant on an artificial diet; this may be due to too small quantities of food or too weak food mixtures, leading to insufficient residue to form a normal amount of feces. Infants fed on cow's milk mixtures, especially when insufficient sugar is added, will in time pass soap stools which are dry and putty-like and alkaline in reaction, with a resulting constipation. This error in feeding will be given

further consideration under the title of Overfeeding with cow's milk with insufficient carbohydrates. Infants fed on diet low in fat, although it may contain a sufficient amount of carbohydrates and protein, frequently have an accompanying constipation due to the fact that the carbohydrates and protein are almost completely absorbed and little residue remains. A considerable portion of the fat ingested in high-fat diets is excreted in the feces as fatty acids, neutral fat and fat soaps. Excessive quantities of slowly fermentable carbohydrates, such as starch, will lead to constipation, unless accompanied by sufficient sugar which by its more rapid fermentation causes active peristalsis. From the foregoing a conclusion may be drawn that the proper balancing of a diet, as discussed under infant feeding, is of importance in the prevention of constipation.

The boiling of milk may be the cause of constipation, in large part due to the fact that the breaking of the curd leads to more rapid digestion and secondly due to the fact that these smaller curds result in less irritation of the intestinal wall. Both of these factors result in more complete absorption of the intestinal content. It should be remembered that the average infant which is thriving on its diet and making normal progress, even though it is passing "soap stools," is not in need of radical dietetic changes. More commonly a simple readjustment of the food ingredients will result in a change in the character of the feces. Only a careful study of the physical development of the infant, which should include examination of the blood, urine and osseous system, should lead one to the conclusion that the constipation is of pathological significance, due to an improperly constituted diet.

Diagnosis. Every effort should be made to discover the cause and the principal seat of the constipation. To arrive at the cause, the infant's age must be given consideration and a careful study must be made of its diet,

habits and constitution. Most of the non-inflammatory obstructions are seen in the first days or months of life, and are equally common in the breast-fed. Whether due to obstruction or diet, it is important to determine whether the seat of the trouble is in the small intestine, colon or rectum. In both the breast- and bottle-fed infants, if local means, such as suppository or enema, will result in rapid passing of a normal stool, the fault will usually be found in the rectum, which is in need of increased stimulus to assist .it in emptying. This is especially common in breast-fed infants, as well as artificially fed, who are too young to make use voluntarily of the abdominal wall. These cases are usually unaccompanied by other symptoms, unless the stool be hard and dry, which may result in injury to the anus. In the cases in which an improper diet is .the underlying factor, the stools are usually pathological and are associated with evidences of discomfort, flatulence, irritability and insufficient gain in weight. Cerebral disease, as well as constitutional inferiority, as seen in cretinism, rickets, athrepsia and other systemic conditions must be properly interpreted.

Treatment. In breast-fed babies, and not. infrequently in infants fed on boiled milk, we frequently find a sluggish rectum, which is evacuated to better advantage by the use of simple mechanical means than by the use of physics. A lubricated catheter, a simple suppository made from glycerine or soap, or one or two ounces of a saline enema or sweet oil injection, can be recommended. If properly used, they are not harmful, nor do they create bad habits which are often ascribed to them. A regular hour for their use, with proper training, creates regular habits, and in most instances the condition improves to such an extent that they can be discontinued. Most infants can be trained to regular evacuations by the fourth or fifth month. The infant should be well supported on the mother's lap, over a

chamber, which she may hold between her knees. This is done to best advantage after a feeding or just before the morning bath, and a suppository may be used until the infant realizes that the operation is undertaken for a purpose. Abdominal massage, instituted at a regular time, is of great assistance in promoting the emptying of the lower bowel. In older infants this may be combined with muscle exercises.

The treatment of the anomalies of the gastro-intestinal tract causing constipation is usually surgical.

Drugs, except for temporary use, should be given the last consideration in the treatment of constipation. The stronger cathartics, such as salines, castor oil or calomel, are to be avoided. When the dietetic and local measures fail, the addition of one or two teaspoonfuls of milk of magnesia (Magma magnesiæ N. F.) to the day's feeding, or the administration of mineral oil in doses suitable to the infant's age may become necessary.

Constitutional conditions must be corrected by proper treatment.

Dietetic Treatment. In the breast-fed infant usually no change in the diet is indicated, unless it is insufficient in amount. In the artificially fed infant a change in the character of the food may be all that is necessary to overcome even protracted constipation.

In the presence of soap stools in infants who are thriving, usually a reduction in the amount of whole milk or substitution in part with skimmed milk temporarily and an increase in the amount of sugar will often change the character of the stool sufficiently to relieve the accompanying constipation. It should, however, be remembered that the so-called soap stools are most frequently seen in the artificially fed infant on high milk mixtures, and that it is a common finding in infants who are making excellent progress.

Under such circumstances radical dietetic changes are not indicated. And it should be remembered that in an

attempt to overcome this type of constipation great injury may be done, if the fat and protein content in the diet are reduced excessively. The use of local means to assist in emptying of the rectum is therefore to be advised rather than the institution of extreme changes in the diet.

When the feeding of high milk mixtures with an insufficiency of carbohydrates has resulted in systemic changes associated with loss of turgor, stationary weight and metabolic disturbances, a radical change in the diet is indicated, and this will be treated more in detail under the heading, Overfeeding with cow's milk mixtures with insufficient carbohydrates.

In the presence of constipation, where the maltose-dextrin compounds have been used, a change to milk-sugar or cane-sugar, or one of the maltose-dextrin compounds containing a high percentage of maltose and potassium carbonate is often indicated. Occasionally the addition of cereal water to the diet is of benefit. Those made from whole cereal are more valuable than when prepared from the dextrinized flour. The addition of from one to three tablespoonfuls of the dry or liquid malt-soup extracts added to the day's feeding, is frequently all that is necessary to relieve constipation. In infants where constipation is distressing and other dietetic changes fail, a week or two on Keller's malt soup will relieve the condition. (See Appendix.)

When the infant is old enough constipation is best relieved by the addition of vegetable soup or vegetable and fruit purées.

4. Abnormal Stools.

Number. Breast-fed infants usually have one to four stools a day. Bottle-fed infants have one or two, and infants fed on high milk and low carbohydrate diet usually require some local measure to induce even one. The fewer the stools passed the greater is the likelihood that

they will be constipated. Increased peristalsis and secretion tend to increase the number of stools. Strong acid or alkaline bases tend to irritate the mucous membrane, more especially of the large intestine, with resulting loose stools. These types of stools also have a tendency to irritate the buttocks if not removed shortly after being passed. The hard casein curds, seen when an excess of raw milk is fed, also tend to increase peristalsis. Frequent stools are usually associated with an increase in mucus, the same factors causing both conditions.

Reaction. *Tests.* Blue and red litmus paper may be used to test the reaction. It may be necessary to moisten the stool before making the test by adding a few drops of water. The central portion of the stool should be used for making the test in order to avoid its being affected by urine. Breast-milk stools are almost always acid. Those of the artificially fed are acid if the fat and carbohydrate in the diet outweigh the protein. If the reverse is true they may be alkaline. This is especially common with the skim lactic acid or sweet milk mixtures with low sugar or fat. In the majority of instances stools when well digested will be found to give a neutral reaction and will not affect the litmus paper.

Color. Breast-milk stools are usually yellow to orange.

Green Stools. More especially when frequent in number they may pass with a greenish tinge, due to the fact the biliverdin is passed before it can be reduced, or they may be passed as yellow stools and turn green after passage. Green stools in an infant making good progress have no great significance. In the artificially fed, more especially, the soap stools tend to turn green upon standing, due to the oxidation of the bile salts from bilirubin to biliverdin. This is especially true in the presence of moisture, as from being saturated with urine.

High protein stools have a tendency to vary from gray to olive green and the green color is compatible

with normal health and unless the infant is not thriving should not lead to radical change in the diet.

In the artificially fed infant there is a tendency for the stools to assume a gray, putty-like appearance after the first few weeks on an artificial diet consisting largely of cow's milk and sucrose or lactose. Malt sugars, when fed in moderately large quantities, tend to give the stools a brownish tinge.

Gray stools are those in which the bile salts have been reduced in a large part to urobilin, the biliverdin being practically absent. This is characteristic of the so-called soap stools seen in high milk and low carbohydrate feeding.

Brown stools may vary from a light to a deep brown in color. In most instances this is due to feeding with the various maltose-dextrin compounds. Deep brown color is especially characteristic of feeding with Keller's malt soup and when extract of malt is added to the milk mixtures to overcome constipation. The color will vary directly with the amount of malt added. The cereal waters, when added in considerable amount or strength, tend to tinge the stools brown. In older infants meat, meat juices, some of the vegetables, and iron given in the form of medication will color the stools.

Pink Stools. Urates from the urine may cause a pink color around the edge of the feces. This may also occur through oxidation of the bile salts or through the action of the alkali in the napkin on urine or stool. The latter conditions are, however, exceptional.

Dark Stools. Meconium may vary from green to dark brown or even black. Starvation stools, consisting largely of mucus and containing little fat residue, are usually dark brown in color, due to the pure bile salts contained. The drugs which are most frequently administered and cause dark stools are iron, bismuth, argyrol, and charcoal.

Bloody Stools. The influence of blood on the color of the stool depends upon the amount and time of contact with the intestinal contents. Only blood which is passed shortly after hemorrhage retains its bright red color. Old blood gives the stool a tarry appearance. In the new-born it is often difficult to differentiate this from normal meconium. This should be borne in mind in considering the possibility of the presence of hemorrhagic disease in the new-born. In infectious diarrhea blood is frequently seen in stools, the result of hemorrhage from ulceration in the wall of the colon and appears as specks or masses and is more often of bright red color. These cases usually show an excess of mucus as well. Smaller blood masses may occur in the presence of polyps and fissures in the rectum and anus. In intussusception blood and mucus form the basis of the stool and after the first movement there is little fecal matter.

Odor. Breast-milk stools usually smell sour. Diets containing large amounts of carbohydrates in proportion to the milk contained have a more or less marked tendency to a sour odor. This is especially true in feeding with Keller's malt soup. The soap stool and the stools of infants fed upon high proteins have a very foul odor. The starvation stool has a peculiar musty odor, due to an excess of mucus. This stool is characteristically seen in under-fed infants and in those on starvation diet. Butyric and lactic acids, when present in considerable amounts, can be detected by their odor. The odor of decomposing urine, more particularly when there is a marked ammoniacal odor, will cover up the odor of the feces. Therefore, to determine the fecal odor the stool should be examined shortly after it is passed.

Characteristic Types of Stools. *Starvation stools* are usually greenish brown or brown in color, contain little fecal matter, are composed mainly of mucus and have a tendency to have a moldy or musty odor. They are

most commonly seen in the severe types of vomiting and anorexia or when there is inability to swallow the food, also at the end of the starvation period in the treatment of diarrheal disturbances. Care should be taken so that the starvation stools will be differentiated from the meconium in the new-born, otherwise nursing on dry breasts may be overlooked. They must also be differentiated from stools containing decomposed blood.

Curdy Stools. Curds are seen as undigested masses, and may be formed from fat or protein, or a combination of the two.

Fat curds are far more common than protein curds, and are usually seen as small, soft, whitish or yellow masses, either sprinkled throughout the stools or not infrequently making up a large part of the stool. They are usually intermixed with mucus, which is present in excess; in fact, most of the curds are completely surrounded by mucus which interferes with its digestion. The chemical composition can easily be demonstrated by the usual tests for fat. Breast-fed infants very commonly show curds of this type, and usually they have very little pathological significance in these infants. The mucus is probably secreted in large amounts because of the irritation caused by the presence of fatty acids. Mistakes are often made in the interpretation of this last group of stools. Because they are full of fat curds it is supposed there must be a fat indigestion. The curds are the *result of indigestion or constipation, rather than the cause.*

Casein curds are far less frequent, and present quite a different appearance. They are seen only in the presence of feeding with *raw* milk.[1] They appear as smooth, hard masses, of a yellowish-brown color, with white center when broken, and are usually larger than the fat curds. They are also fewer in number, and may be

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[1] Brenneman, J.: Archives of Pediatrics, xxxiv, 81, 1917.

found mixed in feces which otherwise appears normal. The addition of ether, which causes the fat curds to go into solution, results in hardening and toughening of the protein curds. This is an easy method of differentiation. Such stools have usually an offensive odor.

The *fat curds,* if numerous, call for a considerable reduction in the fat percentage. The *protein curds,* if numerous and persistent, should lead one to reduce the milk, at least temporarily, or to boiling or citrating the milk, which causes their disappearance. In a dyspeptic infant on a high sugar diet with hard curds in the stools, reducing the sugar from the raw milk mixture, thereby lessening the frequency of stools and slowing peristalsis, may cause the hard curds to disappear—that is, the sugar diarrhea which prevented digestion of the casein has been remedied.

Neutral fat is rarely present in stools, and when found is often indicative of fat intolerance. In most instances when it is found, it is proved later to be not from the milk but from castor oil, olive oil, or some ointment used on the baby. Fatty acids are not uncommonly found. Breast milk stools contain them frequently and are not considered pathologic. When found in cow's milk stools, it signifies impaired fat absorption. Stools containing free fatty acid globules almost always contain also many fat curds and a great deal of mucus. In considering fatty acids it must be remembered that formic, acetic, butyric, lactic, succinic acid, etc., are fatty acids or derivatives of them, as well as stearic and oleic and palmitic acid. Formic acid, acetic acid, etc., are lower in the series, more irritating to the mucous membrane, and soluble in the watery content of the stool, not appearing on microscopic examination. Stearic, oleic and palmitic acids are very complex and high in the series, are insoluble in water, presenting themselves as oily, colorless globules, and are easily distinguishable as red or orange globules when stained with Sudan III.

In examining a stool for fatty acids, we look only for globules. Finding none, we presume that that particular phase of fat indigestion is not present. But the stool may be very acid from the presence of fatty acids lower in the series that do not form globules but are in solution, and clinically it makes little difference whether the higher or lower fatty acids are present.

Soap Stools. These are light in color, large and dry, and do not adhere to the napkin. They are seen in feedings in which cream or whole cow's milk is in the excess as related to the carbohydrate content.

Grover[1] says that the normal soap stool contains soap and protein matter, the proportion varying with the relative amount of fat and protein in the food. If the fat in the formula is high and the protein low, we shall get a typical soap stool, provided there is no indigestion. Such a soap stool is formed and of very light color. They are usually dry and constipated. Soap stools rarely number more than two a day. They are alkaline in reaction, sometimes almost neutral. When spread out they appear smooth and dull. On microscopic examination, no neutral fat or fatty acids are found; but on being heated with acetic acid, almost every particle of solid matter is found to be changed to globules of fatty acids, formed from the soaps. As the protein in the food is increased and the fat decreased, there will be found more and more solid matter on the slide, that will not break down with heat and acetic acid. This raises the question of the value of the microscopic examination of the stool for soaps. It has usually been considered that when the microscopic field was "loaded" with globules, *i.e.,* virtually all the solid matter changed to globules of fatty acids, the baby was not taking care of the fat very well, or might be on the edge of an acute exacerbation of a chronic fat intolerance. If the baby

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[1] Grover, Joseph I,: Jour. A. M. A., 1921, 76, 365.

were taking a formula composed of 3 per cent. of fat
and 1 per cent. of protein, we should expect to find lit-
tle else besides soaps in the stool. If the fat in the food
should be kept at 3 per cent. and the protein raised to
2.5 per cent., we should get a different picture micro-
scopically. We should find that only one-third or one-
half of the solid matter was changed to globules, the
remaining solid matter being derived from the protein
in the food. Just as much soap will be passed in the
day, but on microscopic examination it will seem much
less because it is scattered and separated by the increased
amount of protein matter. Microscopic examination of
a stool for soaps, without considering the formula, is
just as misleading as trying to estimate the red count in
a fresh blood smear by comparison with the white cells
without first having made a white count.

Boiled High Casein, Low Fat Mixtures. These stools
are most typically produced when any fat-free milk,
which has been boiled from three to five minutes, is
fed in amounts over two ounces per pound of body
weight and in the presence of minimum or moderate
amounts of sugar. Their consistency may be soft but
more frequently they are quite solid and have a tendency
to dry out rapidly. They usually have a greenish tinge,
shading to light brown. When fat-free lactic acid milk
is used in place of sweet skim milk they are somewhat
more of a brownish shade. Grover has called attention
to the very characteristic shiny surface produced when
a tongue depressor or spatula is passed through it. This
sheen must be differentiated from the glistening appear-
ance of mucus and also from the natural moisture of all
normally passed stools, due to their watery content. The
high protein stool is dull on the outside, where it is dry
from contact with the napkin and in this respect greatly
resembles the soap stools.

As the fat in the diet is increased the greenish color
and transparency soon disappear and with further in-

crease in the fat the smoothness and gloss are lost, and when the fat outweighs the protein the dull soap stool appears. The high casein stool described should be considered a normal one when feeding large quantities of boiled skim milk. As stated, the characteristic appearance changes directly with the amount of carbohydrate and fat added.

In the presence of an excess of fat and protein in the stool, upon heating with acetic acid and then staining with Sudan III, the soaps are changed into fatty acid globules, while the protein is seen as solid matter that is not affected by the heat and acid.

Carbohydrate Stools. Starches are readily stained with dilute tincture of iodine or Lugol's solution. The particles of undigested starch stain blue or black. Stools containing much fermented starch are loose, acid, light brown, and excoriating, and contain much mucus. In fact, these stools are often mistaken for mucus. This type of stool is most likely to be found in babies having indigestion from certain of the starchy proprietary foods. Small, brownish specks are often found in normal stools, representing the indigestible cellulose envelops of cereal foods.

Indigestion of sugar presents no typical stool. Stools resulting from sugar fermentation are frequent, very acid and excoriating, and often watery. The solid parts are usually full of small air bubbles, formed by the chemical decomposition. These may be demonstrated by pressing out some of the stool between a slide and cover glass and examining with the low power. The stools are usually green because of the action of the acids on the bile pigments. Stools from sucrose or lactose fermentation are green, while those from maltose-dextrin preparations are brown.

5. Milk Idiosyncrasy.

A few infants show a true idiosyncrasy to cow's milk, which is overcome only with great difficulty, even when the milk is carefully modified. The true cause of this condition is still in dispute. However, it may be said that some of these cases are undoubtedly due to anaphylaxis. On the other hand, some of them are undoubtedly not explained on this basis. Infants suffering from such idiosyncrasy will usually refuse the milk, and when it is forced upon them it results in vomiting, diarrhea, and frequently an urticario-erythematous rash. Cow's milk feeding in these cases is often associated with a low-grade fever. The symptoms speedily subside upon the administration of castor oil and the withdrawal of milk. This class of cases offers great difficulty in feeding during the first year of life, as carbohydrates must necessarily form a considerable portion of their diet. Broths, cooked cereals, and vegetable purées should be gradually added to the diet as soon as they can be digested.

Replacing the cow's milk in the diet by goat's milk will, in many cases, relieve all symptoms.

CHAPTER II.

GENERAL CONSIDERATION OF NUTRITIONAL DISTURBANCES.

Evolution of the Conception of Nutritional Disturbances.

OUR ideas on this subject have undergone considerable change during the past few years. Older authors viewed the nutritional disturbances as conditions limited to the stomach and bowel, and likened them to similar conditions in the adult, with the exception that more serious results were to be expected in the infant because of the slight physiological resistance, the infant's body being more favorable to a severer course.

For many years the classification of Widerhofer, of the Vienna school, first published in 1880, and based on an anatomico-pathological basis was the one in general use. These conditions he grouped as follows:

1. Functional disturbances, as acute and chronic dyspepsias.

2. Enterocatarrhs, with more or less marked histological changes and clinical findings.

3. Follicular enteritis, with deep-seated inflammatory and ulcerative changes, especially in the large intestine.

4. Cholera infantum (this latter, a severe type of enterocatarrh, was classed as a distinct clinical entity).

Clinical observation soon convinces one that the cases do not follow the distinct types in the above classification, mixed and progressive types being the rule. In many instances far-reaching after-effects remain, and, again, in others of the severest types few if any anatomical lesions are demonstrable at autopsy. Especially in young infants we find marked and often general disturbances following in the wake of what seemingly were localized gas-

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tro-intestinal lesions, with the result that the systemic and not the intestinal symptoms were of more serious impòrt. Again, we know that many findings formerly attributed to invasion of bacteria or their toxins can now be attributed directly to improper metabolism of the food ingested.

Food Injuries. The nomenclature covering this subject has also changed, and we now adopt the term "Nutritional Disturbances" in place of "Gastro-intestinal Diseases," the former covering the functional and anatomical disturbances, as well as the bacterial and food traumas. It is, however, necessary in order to justify the newer nomenclature to look upon nutritional disturbances not as localized in the gastro-intestinal canal, but as general affections involving the whole organism in one of the most vital of its functions. The gastro-intestinal symptoms form only a part of the clinical picture; therefore, in its fullest conception the mental state, weight disturbances, changes in the temperature, pulse, respiration, etc., may become as important in their interpretation as the diarrhea. Two schools of pediatrics have given us the nucleus for our present views on nutritional disturbances and their classification—those of Czerny and Finkelstein. Czerny's work antedated that of Finkelstein by several years, and he based his classification on what he considered injuries due to overfeeding with individual food elements. These he called "food injuries," and described them as due to fat, starch, sugar, protein, and salts, individually or in combination, either when given in excess, or when given to an infant with lowered tolerance for these food elements.

Finkelstein viewed the nutritional disorders from a broader standpoint. He considered them *"as the gradual development of an increasing intolerance for food"*— step by step, from the mildest disturbances, in which the only striking symptom is failure to gain in weight, through the severer diarrheas, up to the final stage of

intoxication, when the infant is in a state of *"metabolic bankruptcy."* In his classification we see one increasing process, the important factor of which is found in the fact that the infant can tolerate less and less food, until finally any food in any amount acts harmfully. The stages of the various disorders under the Finkelstein classification must therefore necessarily merge gradually into one another, and lack in definiteness, and at times present a picture so complicated that an exact diagnosis as to the stage be temporarily impossible.

The schools of Czerny and Finkelstein laid the practical foundation for the combined etiologic and clinical classifications which form the working basis for our present grouping of nutritional disturbances. The nomenclature of Finkelstein applied to the groups in his clinical classification, namely Disturbed Metabolic Balance, Dyspepsia, Decomposition and Intoxication, except for the latter, give the clinician very little insight into the underlying etiologic causes. In the first two editions of this book, the Finkelstein terminology was used, but in this third edition the more descriptive terms.

Overfeeding with Cow's Milk with Insufficient Carbohydrates replaces the *Disturbed Metabolic Balance.*

Nutritional Disturbances Characterized by Diarrhea replaces *Dyspepsia.*

Athrepsia and Marasmus replace *Decomposition.*

Anhydremic Intoxication replaces *Intoxication.*

Before entering upon a general discussion, it may be wise to review some of the theories promulgated for the advantages of human over cow's milk in infant feeding. Biedert believed that the decomposition products of protein digestion were the important factors. This idea has not been substantiated clinically. Hamburger advanced the idea that the albumins foreign to the human body contained in cow's milk were important. factors. This is true in a limited number of cases of milk idiosyncrasy. Czerny believed that the fat, and, again, the sugar, are

the important factors. L. F. Meyer believed that the whey content, and more especially the high salt content of whey (0.75 per cent. as compared with 0.2 per cent. in human milk), predisposed to intestinal injury, following which trauma, fats and sugars play an important part. Marfan, Escherich, Pfaundler, and others believed that specific protective bodies of unknown nature were contained in raw human milk, which are of vast importance as immunizing bodies.

Etiology. In the proper consideration of the nutritional disturbances we must consider the whole organism rather than an individual organ or system of organs, in formulating our diagnosis and outlining a course for treatment. In past years the tendency has been to consider the gastro-intestinal disturbances as conditions limited to these organs without due consideration of the systemic involvement, both preceding and following in the wake of traumata which primarily affected the digestive tract. It is also to be remembered that when we have an involvement of the gastro-intestinal tract, that only rarely is the underlying pathological condition limited to a single segment, but that much more frequently the entire tract, including as well the accessory digestive organs, are likely to be involved.

In the study of a given case it is first necessary to decide, if possible, as to whether it is a mild or a serious disturbance. This is of greatest importance from both the standpoint of prognosis and treatment.

To form a definite conclusion we must consider the age of the infant, realizing that the age at which the infant becomes sick is of great importance, the previous history of the infant as to diet and preceding nutritional disturbances and infections, together with a careful study of any existing constitutional anomalies or idiosyncrasies. In the consideration of the latter the family history as to serious nervous diseases, tuberculous infections, and the history of the result of previous

pregnancies, are of extreme importance. Full consideration must be given to the surroundings under which the nutritional disturbance developed. Unhygienic conditions in the home are of the greatest importance. It is also well known that a greater tendency exists to development of this class of disturbances during long continued hospital and institutional confinement because of exposure to infection and lack of exercise. The season of the year should be considered in arriving at a conclusion as to the underlying etiology.

According to the school affiliation of the physician, one speaks of food injuries, basing his classification upon the etiological factor, while the other classifies them in the clinical light. Almost every year brings with itself new modifications in the presentation of the subject. Many of them do not lead to the goal of the practical.

The clinical pictures which the nutritional disturbances of infants assume in their course are, to a certain extent, uniform, and in fact they can usually be included in certain definite types. On the one hand are the acute and subacute disturbances, of which the most important symptoms are related to the gastro-intestinal canal, especially diarrhea with a tendency to more or less general disturbance, more particularly of the nervous system. In many of the chronic disturbances, manifestations on the part of the digestive tract may be entirely absent, and they almost never present a characteristic impression of the primary etiological factor. All of them have in common, signs of lowered immunity, abnormal behavior of the weight curve, alterations of the color and turgor of the skin. In fact the differences are likely to be quantitative only.

Thus, in both acute and chronic disturbances there are often very narrow limitations for the etiological diagnosis, based upon clinical findings. And it is often difficult to determine whether alimentary causes due to over- or underfeeding, recurrent infections or constitu-

tional inferiority are the underlying factors. Knowing these limitations the importance of a carefully taken history cannot be overestimated. The history of the prodromes, onset and development will frequently give the data necessary to differentiate between the primary alimentary disturbances and those secondary to infections and constitutional anomalies.

All of the etiological possibilities are exhausted by (1) alimentation, (2) infection, (3) constitution, and (4) environment (weather and hygiene). Their sharp differentiation, however, is often difficult.

And while a proper estimation of the degree of the nutritional disturbance may be gained from the history (determination of the preceding illnesses and the determination of the development history of the present illness), a detailed study of the actual picture, as presented in the individual infant, as met by the physician in his daily practice, must form the basis for properly outlining the course of treatment.

Even in those nutritional disturbances in which the etiological diagnosis is not only probable, but positive, the treatment is not based upon the etiology, but rather upon condition of the individual infant at the time of examination. Thus, if proper treatment of a case presenting diarrhea as the most marked symptom, was possible only after we had the knowledge of the factors, which produced the diarrhea, the institution of proper therapy would in many instances, of necessity, be delayed.

On the other hand, there are diseases, although symptomatically almost completely alike, varying widely in their course and response to the same treatment and in their final outcome. In other words, the individuality of the infant is often the deciding factor in the prognosis of the case.

By exact clinical analysis we are able in most cases to arrive at a proper estimation of the degree of the nutritional disturbance and thus to the institution of the

plan of treatment, and this object may be reached in almost every case by a study of the preceding history of the infant and by a consideration of the progress of the disturbance.

We know that the bacteria and their products as encountered in the food administered, are less often the offending factors than formerly supposed, and that improper food, either quantitatively or qualitatively, is of equal or greater importance in the causation of nutritional disturbances. To avoid confusion in our discussion of this vast field, we will first consider food injuries and discuss the infections incidentally, as they affect the former, and at a later period discuss the infections in their relation to the nutritional disturbances more in detail.

From the foregoing it becomes evident that to discuss the whole subject properly it becomes necessary to consider them from both the etiological and the clinical standpoints.

ETIOLOGICAL CLASSIFICATION OF NUTRITIONAL DISTURBANCES.

1. Overfeeding with Milk Mixtures, of:
    (a) Correct composition (too frequent or too much).
    (b) Incorrect composition:
        (1) With insufficient sugars.
        (2) With excess of fat, protein, sugar or salt.
    (c) Raw milk (with resulting mechanical irritation due to large, hard protein curds).

2. Underfeeding with Milk Mixtures of Correct Composition.

3. Underfeeding with Diets of Incorrect Composition.
    (a) Diets with insufficient milk and sufficient sugars.

(b) Diets composed chiefly or entirely of starches.

(c) Diets low in vitamines.

4. *Feeding with Spoiled Milk (Decomposition Products of Milk and Bacterial Toxins).*

5. *Subnormal Food Tolerance.*

(a) Preceding dietetic errors and nutritional disturbances.

(b) Infections:

(1) Enteral: dysentery, typhoid, etc.

(2) Parenteral: otitis, pharyngitis, pneumonia, pyelitis, etc.

(c) Extremes of temperature (heat of summer and cold of winter).

(d) Improper hygienic conditions and "hospitalism."

(e) Constitutional anomalies:

(1) Organic (pyloric stenosis, megacolon).

(2) Functional (idiosyncrasy to cow's milk. Exudative diathesis. Neuropathic diathesis.)

Nutritional Disturbances Due to Overfeeding. This is one of the most important of all etiological factors. The disturbance may be due to a diet of correct composition, but quantitatively too great for the individual case, or a diet with an excessive amount of one or more constituent ingredients.

Nutritional Disturbances Following Underfeeding. We recognize two types: (1) Qualitative, and (2) quantitative. Sooner or later the results are similar. The former diets, *qualitatively* wrong, are frequently seen where the caloric requirements are met, but one or more of the necessary food elements is insufficient. An example of this is seen in the feeding of carbohydrate rich food, as condensed milk, malted milk, etc. When the minimum requirements for growth and development, at least for both organic and inorganic salts, are met in such

a diet, the organism may be able to overcome the excess of one ingredient, but if this is not true, sooner or later some grave complications will result. With diets composed largely of cereals or cereal waters, as is frequently seen when these are used to replace the milk mixtures, in the course of diarrheal disturbances, the clinical picture of inanition develops with great rapidity. When we feed less than a sustaining diet of 32 calories per pound body weight or 70 calories per kilogram, there soon results a *quantitative* inanition with all its undesirable after effects. More recently we have learned to classify infants fed for prolonged periods on diets low in vitamines among those suffering from qualitative inanition. To judge such errors in diet, each individual infant must be studied as a distinct entity.

Nutritional disturbances following feeding with spoiled milk may be due to decomposition products of milk, bacterial toxins or pathogenic organisms contained in the milk which may result in secondary enteral or parenteral infections.

Nutritional Disturbances Due to Subnormal Food Tolerance. Many factors can cause such a state of affairs. The history of preceding dietetic errors and nutritional disturbances should be given careful consideration. Thus, *e.g.*, diarrheal conditions, due to a given cause, will in the normal infant give a far better prognosis than in the infant which has suffered from over- or underfeeding, while in the athreptic infant even moderate diarrheal disturbance should give grave concern.

Infections, enteral and parenteral will show marked individual differences in producing a subnormal food tolerance. The natural immunity of the healthy breast-fed infant affords the best example of the importance of a diet in the establishment of resistance to infection. The susceptibility to infection is increased by every nutritional disturbance as well as by constitutional inferiority.

Most of the disturbances due to *extremes of tempera-
ture* are noted in summer, during which season they are
not only due to the effect of temperature upon the infant,
but also due to spoiled food. Exposure to cold is often
an etiological factor through lowering of immunity.

Improper hygienic conditions in the home predis-
pose to impairment of digestive functions as well as to
secondary infections. In this connection it should be
remembered that long continued hospitalization, even in
the presence of good surroundings and proper diet, may
result in lowered food tolerance due to lack of "mother-
ing" and exposure to infection.

Constitutional anomalies may result in various types
of nutritional disturbances, depending upon their nature.
In the presence of hypertrophic pyloric stenosis, quan-
titative inanition, with resulting athrepsia, is likely to
develop in uncorrected cases. In the presence of intes-
tinal stasis, due to a weakened intestinal musculature,
as seen in megacolon, and in the course of severe rickets,
a marked constipation develops, with retention of the
intestinal contents for a prolonged period, both in the
ileum and large intestine, with resulting absorption of
the products of decomposition. Fortunately few infants
show a true idiosyncrasy to cow's milk. This causes very
great difficulty in feeding young infants in the absence
of a supply of breast milk. The intolerance of infants
suffering from exudative diathesis, to overfeeding as
well as the tendency of neuropathic infants to develop
exaggerated reflex action in the gastro-intestinal tract,
with resulting cardio- or pylorospasm, and increased
peristalsis of the intestinal tract with the development
of colic and diarrhea, offer some of the most difficult
problems in infant feeding. This group of cases calls
for repeated observation and study of the individual
infant.

General Symptomatology.

The normal healthy infant, with a well-balanced metabolism, reacts to food as follows:

1. An elastic, pink skin, a well-developed panniculus adiposus, well-colored mucous membrane.   Its tissues should feel firm.

2. One should expect certain muscle and bone development according to the age of the infant.

3. A uniform rectal temperature (98 to 99 degrees F.), almost a monothermia.   Any considerable deviation is abnormal.

4. It should show a regular, steady gain in weight.

5. The bowel movements should be regular, and should vary with the food ingested.

6. Its disposition should be happy, and its nervous functions normal.   It should sleep well, and be satisfied with feedings at three- to four-hour intervals.

7. It should show a wide tolerance for food, both as to the diet as a whole, and to the individual food elements.

8. Renal, circulatory and respiratory functions should be normal.

Bearing in mind the attributes of the healthy infant, we are now in a position to review the factors leading to and influencing our present conceptions of the nutritional disturbances, based on an ascending series of pathological stages in those infants whose tolerance for food has been overstepped, either because of overfeeding or because of diminished or abnormal tolerance on the part of the baby itself.

The clinical observation soon convinces one that while simple types are not uncommon, mixed and progressive types are of frequent occurrence.   Exact observation of variations in the general condition of the infant and the correct interpretation are necessary for a proper understanding of the clinical symptoms.   A correct diagnosis

of a condition can usually be made from the clinical analysis of the symptoms with the aid of the previous history. In order to outline the proper plan of treatment, it is necessary to decide whether in a case under observation there is a mild or a serious disturbance. This is of the greatest importance in the choice of good mixtures, both in qualitative and also in quantitative respect. The physician needs to learn that the condition of the infant should not be estimated by any single clinical symptom, but rather according to the effect upon the general condition of the patient. He must take into consideration the weight curve, the color of the skin, the muscle tone and turgor, the presence of anemia, the state of the sensorium, and the condition of the stools, and in addition to these any other symptoms which may develop. As an example, he should not consider only the presence of diarrhea, but even more important, the reaction of the infant to the loss of body fluids and salts through excessive stools. He should remember that age is an important factor and that nutritional disturbances in the first three months of life should always be regarded as serious, in other words, a diarrheal condition in the first six weeks of life is far more serious than the one developing in the sixth month of life. The disturbance in the infant whose history shows that there is nothing defective in his constitution, allows of a better prognosis than would be expected in a weakling. By exact clinical analysis we are able in most cases to arrive at a proper estimation of the degree of nutritional disturbance and thus to institute a plan for treatment.

Clinical Classification of Nutritional Disturbances.

In the clinical pictures that may develop on the basis of the above-mentioned etiology, several more or less distinct types or syndromes may be differentiated. These

may be dependent primarily or exclusively upon the dietetic errors, or they may be influenced by secondary etiological factors. The degree of reaction depends upon the extent to which the infant's metabolism is disturbed.

For practical purposes of diagnosis and treatment the nutrition disturbances may be grouped on a clinical basis, as follows:

1. Nutritional Disturbances Unassociated with Diarrhea.

2. Nutritional Disturbances Characterized by Diarrhea. (Diarrheal disturbances.)

3. Athrepsia (Marasmus. Decomposition).

4. Anhydremia (Anhydremic intoxication. Intoxication.)

These types represent stages in the course of nutritional disturbances and one form may lead rapidly into another, if the errors in the diet are not remedied, or when secondary complications, such as infections, arise.

CHAPTER III.

NUTRITIONAL DISTURBANCES UNASSOCIATED WITH DIARRHEA.

Overfeeding with Cow's Milk with Insufficient Carbohydrates.[1]

THE syndrome to be described under this cause appears in the pediatric literature under various synonyms. Among them are the following:

Disturbed metabolic balance, weight disturbance, malnutrition, hypothrepsia (Parrott), hypotrophy (Langstein), fat constipation, bilanzstoerung (Finkelstein), milchnahrschaden (Czerny-Keller).

This nutritional disturbance is due to administration of cow's milk mixtures in which the error is based upon an excess of cream or milk in the diet in the presence of an insufficiency of sugar. A disturbance in the metabolic balance is rarely seen in the previously normal infant until the amount of cow's milk fed is in excess of two ounces (60 mils) per pound body weight. With this amount of milk the average requirement in sugar to be added will be one-tenth ounce (3.0 Gm.) for each pound of the infant's weight.[2] The clinical signs develop slowly in most cases and follow a period of good progress which ceases more or less abruptly. Among the early signs are a general retardation of development, associated with a loss of body turgor and stationary weight. The severity of the clinical picture will depend upon the ability of the individual infant to

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[1] In the first and second editions these cases were described under the title of Disturbed Metabolic Balance.

[2] It should be remembered that a well-balanced 24 hour diet for an infant should rarely contain more than one quart (1000 mils) of cow's milk and from 1½ to 2 ounces of sugar (45 to 60 Gm.). Further nutritional requirements are to be met by the addition to the diet of foods other than milk and sugar.

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overcome the ill effects of the one-sided diet and this will vary directly with the degree of the error and the time over which the diet has been administered. The diagnosis must be based on the presence of symptoms resulting in definite injury to the organism. Not infrequently the diagnosis is made upon the presence of marked constipation with a passing of soap stools. These are dry and putty-like, have a foul odor and an alkaline reaction. It must be emphasized that the diagnosis of this nutritional disturbance, due to an improperly balanced cow's milk mixture, must not be based upon the presence of soap stools alone. This is the characteristic stool seen in feeding, even when the milk is not in excess of the amount on which the infant will make normal progress.

Etiology. Artificially fed infants are chiefly affected, probably because of the high carbohydrate and low protein content in the breast-milk.

The clinical picture presented by this type of overfeeding, which is due to an improperly balanced diet, is usually seen in infants receiving food mixtures composed of cream and milk, top-milk or whole milk with an insufficiency of carbohydrates, resulting in an improper proportion between the fat and carbohydrates in the diet. In the presence of excessive amounts of carbohydrates we are more likely to see a diarrheal condition. Proteins also play an important rôle in the causation of the clinical picture of this disease, in that in the presence of a relative overfeeding with proteins an alkaline intestinal reaction, necessary to the production of fat-soap stools, is brought about. Not all infants react alike to the same milk mixture, and those who have a low tolerance for milk, as is the case with many of the infants suffering from exudative diathesis, develop the clinical picture much earlier than the average infant. The same may be said of the lowered food tolerance following infections. Fortunately in these infants the tolerance for

carbohydrates is retained and therefore the fat and the protein in the diet can, to a great degree, be replaced by sugar and cereals. It should be remembered that these infants suffer from a carbohydrate poverty or deficiency and therefore in the presence of infections and other complications there is an urgent indication for a high carbohydrate feeding.

Pathogenesis. As soap stools are so frequently regarded as the basic symptom in the diagnosis of this disturbance, their significance will be emphasized. The soap stool must be viewed as an effect and not as the cause. The condition is not due to a fat indigestion, but a disturbance in salt metabolism based on an improper composition of the diet in which there is a relative overfeeding with fat and protein in the presence of insufficient carbohydrates. The stools consist largely of calcium and magnesium soaps formed from the fatty acids. The presence of the relatively large amounts of calcium caseinate in cow's milk leads to an alkaline condition in the intestinal tract which reaction increases the tendency toward their formation. These stools also contain large amounts of calcium phosphate, which in the presence of the alkaline reaction of the intestinal tract are rendered insoluble. The organism may be markedly affected through the lack of absorption of bases and excessive loss of alkalies by increased intestinal secretion. Many infants will overcome the ill-effects for long periods of time without clinical evidence. Others will present evidences of retarded growth, resulting in malnutrition. Rickets may result. The alkalies most involved in the formation of the soap stools which are so commonly seen in this condition are calcium and magnesium. There is, however, also a decreased sodium and potassium retention, as evidenced more especially by increased excretion in the urine. This loss of calcium and magnesium through the stools and the inability to retain sodium and potassium, and the accompanying decrease in water re-

tention, soon lead to weight loss. The soap stools, as stated, contain an excess of calcium and magnesium soaps, and less fatty acids and neutral fats than is seen in the normal stools.

To obtain such a stool, there must be a strong alkaline reaction in the large intestine, and the food elements of the diet are important factors in the production of this reaction.

Fats. An excess of fats in the food leads to an excess of fatty acids in the intestine, with a tendency to the formation of an acid reaction of the intestinal content. To combine with these, alkalies are withdrawn from the body, if insufficient in the intestinal tract.

Proteins, more especially an excess of calcium caseinate, cause secretion of a large quantity of intestinal juice which is alkaline. This in time tends to produce an alkaline intestinal reaction, if not counteracted by excessive fermentation, the former being favorable to the formation of soap stools. In all probability the great calcium content of cow's milk (4 to 1), as compared with breast-milk, also offers another factor in the tendency to formation of calcium soaps.

Carbohydrates. In the presence of sufficient fermentable carbohydrates (disaccharides) in the diet, the intestinal reaction becomes acid, the products of fermentation counteracting the tendency to alkaline reaction, and thus preventing the formation of fat-soap stools.

The decreased absorption of bases and the increased withdrawal of alkalies from the system disturbs the acid-alkaline equilibrium, creating a relative excess of acids. This results in an increased production of ammonia to counteract the loss of alkali in order to prevent acidosis.

We find a striking example of a *paradoxical reaction,* namely, increasing the food (milk or fat) makes the condition worse and causes weight loss, while diminishing the milk with an increase in carbohydrates results in return to normal.

The clinical picture is due to:

1. Excessive withdrawal of salts from the body tissues, due to fat and protein overfeeding.

2. A relative insufficiency of carbohydrates.

3. Bacterial decomposition of food in the intestinal tract.

As previously stated, the fat-soap stools must not form the basis for diagnosis. It must be based upon the study of the feeding history, clinical signs and in some cases· the constitutional anomalies and systemic infections as predisposing causes. On the whole, this clinical syndrome is not as frequently seen as might be expected, because of the ability of the infant to overcome the dietetic error sufficiently to make a fair progress.

Symptoms. There is a retarding of development qualitatively and quantitatively, the infants frequently being undersized, without showing marked general symptoms of disease.

1. *Weight.* Notwithstanding proper or even excessive caloric intake, there may be no gain in weight, or an irregular increase, however, under the normal. (Stationary weight or insufficient gain in the infant corresponds to a loss in weight in the adult. Stationary weight in an infant alone leads to the picture of malnutrition and marasmus.)

2. *Temperature.* Usually we find daily oscillations from 1° to 2°, with a tendency toward subnormal.

3. The child is restless.

4. Sleep is disturbed.

5. The skin is pale, with loss of elasticity and turgor. Intertrigo and eczema are frequently seen.

6. Muscles are soft and flabby.·

7. Regurgitation and vomiting are frequent.

8. Abdomen, tympanitic.

9. *Stools.* In excessive milk feeding the common type is the fat-soap stool, which is foul-smelling, dry, light in color (gray to white), friable, and does not stick to the

napkin. The pale color is due to the reduction of bilirubin to urobilinogen. The odor, in part at least, is due to the decomposition of protein. The large, putty-like masses of stool are moved through the lower intestinal tract with difficulty, even in the presence of active peristalsis. This interferes with emptying of the intestine, the stools becoming very dry through the absorption of moisture due to this long period of retention. Not infrequently their progress through the colon is associated with colicky pains. In the presence of excessive carbohydrates this stool may be lacking, due to the presence of a slight intestinal indigestion. Occasionally bacterial decomposition of the proteins may cause a diarrhea.

10. Immunity is lessened with resulting furunculosis and susceptibility to respiratory, gastro-intestinal, and genito-urinary infections.

11. Urine is usually *ammoniacal,* and contains an excess of sodium and potassium salts. The fact that alkalies, found as soaps, are poorly absorbed from the intestine makes it necessary for the body to produce ammonia to make up for the deficit and to prevent acidosis. This explains the excess of ammonium salts in the urine. The salts are decomposed after passage of the urine by coming in contact with bacteria. The freeing of ammonia by the bacterial decomposition can be prevented by rinsing the napkins after boiling and washing in a $\frac{1}{10,000}$ solution of bichlorid of mercury, the deposited mercurial salt preventing bacterial growth after the urine comes in contact with the napkin.[1]

Diagnosis. Due to the fact that this type of stool with associated constipation is so frequently seen in artificially fed infants, it is difficult to say just when such constipation becomes pathologic. As long as the infant is making satisfactory progress and showing no

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[1] Cooke, J. V.: Etiology and Treatment of Ammonia Dermatitis of the Gluteal Region of Infants, Amer. Jour. Dis. of Children, xxii, 481, 1921.

signs of definite pathology the mere presence of soap stools should be considered as of relatively slight importance and should not lead to radical changes in the diet.

The diagnosis must be based on the clinical picture and feeding history, as follows: Sufficient caloric intake (100 calories per kilogram), with relative excess of fat and protein, and insufficiency of carbohydrates, stationary weight or insufficient gain, loss of turgor, lack of proper development, and usually soap stools, all in the absence of any other causative factor. Underfeeding and all past illnesses which might retard development must be excluded.

Prognosis is very favorable in uncomplicated cases, with a properly instituted diet. In the average case two to three weeks is required to overcome the constipation, and to obtain a gain in weight. Occasionally a severe type is seen which is difficult to overcome, most common in infants with an idiosyncrasy to cow's milk.

Complications. Because of the lowered immunity, infections are common, especially of the nasopharynx, lungs, middle ear and skin and gastro-intestinal and genito-urinary tract. Exudative diathesis is not an uncommon associated condition.

Sequelæ. This condition is often the forerunner of the more serious nutritional disorders, such as diarrhea, athrepsia, and anhydremia. Chronic constipation frequently results, due to the atony of the intestinal wall and abdominal muscles. Rickets frequently develops in these infants.

Treatment. To institute a proper treatment, we must remember that the clinical picture is not dependent on gastro-intestinal findings only, but also on an abnormal intermediary metabolism (therefore the designation Disturbed Metabolic Balance), and that fat overfeeding primarily, and a carbohydrate insufficiency secondarily,

are causative factors, and that protein overfeeding may be an important element.

1. *Diet with Human Milk.* This is by all means the best treatment, especially in young infants. Weight increase may be slow at first, probably due to low salt and protein content of human milk. A loss of more than 6 to 10 ounces over a period of three or four days is frequently seen. More than this should lead one to suspect an error in diagnosis. This loss may be due, as stated, to stopping of a food rich in proteins and salts, and substituting one low in the same. This stage is passed in about four days, when the system adapts itself to the new food ingredients. Temperature and pulse do not change, and the stools assume a breast-milk-stool character. If the stage of reparation is slow, and the child does not gain in weight, the substitution of one meal rich in protein and salts daily will frequently help (buttermilk or skim milk). Mother's milk also helps to increase the immunity.

2. *Diet with Artificial Foods.* In pathogenesis of this condition the milk plays the most important rôle, and this is best counteracted by replacing it in part, with well-tolerated carbohydrates.

(1) In *simple cases* reduce the quantity of milk and add carbohydrates in the form of sugar and starches.

(2) In *severe cases:*

(*a*) Malt soup (Keller's) (p. 437) is exceedingly valuable. Malt soup is indicated in the presence of fat-soap stools which soon become pasty and of mahogany-brown color; the best results with malt soup are obtained in infants from three to six months of age. After four months more milk than given in the original formula must be added to increase the protein content of the diet.

(b) Buttermilk or skim milk mixtures (contain-
ing two carbohydrates, *i.e.*, sugar and
flour). The action of both is the same.
Occasionally it is necessary in young in-
fants to reduce the sugar recommended in
the original formula (see Buttermilk Mix-
ture, p. 436).

(c) Brady's buttermilk mixture No. 1 (p. 436).

Change of the diet is followed by better sleep, im-
proved turgor, skin becomes less pale, less variation in
temperature. Stools change from soap stools to (1) yel-
low-brown, alkaline and fair consistency, when butter-
milk mixtures are fed, (2) acid, softer, mahogany-
brown color when malt soup is fed.

These results of treatment are due to the fact that the
tolerance for carbohydrates is high, and protein toler-
ance is little impaired. Each case should be watched to
see if an excess of carbohydrates is not being given in
the new diet, which is indicated by (a) restlessness, (b)
stopping of weight increase after an early rise, (c) ali-
mentary fever (irregular), (d) too frequent stools. If
the cow's milk mixtures are not well tolerated, human
milk is indicated.

The above mixtures should be gradually replaced by
ordinary milk mixtures after two to eight weeks.

In infants over six months of age one of the most con-
stant and brilliant therapeutic results follows the use
of a limited amount of milk (boiled or citrated) and the
free administration of toast, zwieback, rusk, and cooked
cereals given in increasing quantities up to amounts that
will bring on a steady gain of 6 to 8 ounces a week. To
this diet broth or vegetable soup and orange juice should
be added soon. In other words, *if a baby of six or seven
months does not gain on ordinary milk mixtures, it should
be fed like a normal baby of nine or ten months,* with
the single exception that the milk should be kept rather
low, or at least given cautiously, and preferably boiled

or citrated, or both. In many cases this can be done even in the fifth month.

Underfeeding with Cow's Milk Mixtures of Correct Composition.

The cases of this class include those infants receiving a diet containing a proper proportion of the necessary food ingredients, however, in insufficient quantity. (Too little of a proper food.) These must again be divided into two groups:

(a) *Normal Infants Quantitatively Underfed.* In breast-fed infants this group is more common than in artificially fed. And while in the artificially fed such cases are occasionally seen, this is a far less frequent condition than overfeeding. Because in the normal infant hunger is manifested by crying, restlessness, loss of weight and associated constipation, which fortunately in most instances leads to a proper interpretation, resulting in increase of the diet.

(b) *Infants Suffering from Nutritional Disturbances, Quantitatively Underfed.* These cases are the ones which so frequently suffer from quantitative inanition, due to the fact that the fever, vomiting and diarrhea offer every indication for a reduction in diet, or a starvation diet. While this leads to an improvement in the general symptoms, the remaining hunger stool, because of its greenish-brown color and excess of mucus, is not uncommonly interpreted as a diarrheal stool, leading to prolonged starvation and not infrequently repeated catharsis.

The result of repeated starvation must necessarily be a condition of malnutrition, which sooner or later will result in the stage of athrepsia (marasmus), unless the underlying condition is corrected, and a proper diet instituted.

Treatment. Repeated hunger days and long-continued underfeeding should be instituted only upon defi-

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nite indications, the sudden decrease in the food leading regularly to weight loss and lowered food tolerance.

An initial cathartic is frequently indicated, while repeated catharsis is harmful.

The diet should be as rapidly increased as the infant's condition will tolerate. It should be carefully selected to meet the requirements of the individual infant.

While in mild cases a properly selected diet leads to rapid recovery and gain in weight, in the severe cases we not infrequently see a paradoxical reaction to food, necessitating feeding as described under the chapter on Athrepsia.

In every case the infant's tolerance to food should be carefully studied, and increases made only as tolerance permits.

Hunger stools are rapidly replaced by those of normal consistency in the presence of a proper diet.

Underfeeding with Diets of Incorrect Composition.

(a) Diets with Insufficient Cow's Milk and Sufficient or An Excess of Sugars. This class of cases are most frequently seen in infants fed on proprietary foods, more especially condensed milk and the so-called· "baby foods" composed largely of carbohydrates to which some milk is added.

The tendency on the part of the infant so fed is to store large amounts of fat. They usually appear plump, but have a tendency to develop secondary anemia, rickets and scurvy. Frequently diarrhea will be superimposed in the presence of slight irritation of the gastro-intestinal tract following mechanical or bacterial causes.

Infants fed upon such one-sided diets have a lessened immunity, both to systemic and intestinal bacterial infections. They are therefore liable to recurrent infections.

Treatment. The chief object should be to place them upon a well-balanced milk mixture suitable to their age, to which well-cooked cereals and vegetables are to be added as the age permits.

In changing to cow's milk mixtures from these high carbohydrate diets, it is best to start with small amounts of milk first, adding one ounce of cow's milk for each pound of body weight. This is to be increased as the infant's condition permits. The carbohydrates should be reduced to amounts approximating one-tenth ounce per each pound of body weight, in addition to that contained in the milk. Only in the presence of severe diarrhea should sugars be entirely withdrawn and then with caution, because of the danger of collapse. It is advisable to boil the mixture, at least early in the new feeding, in order to split the casein curds. The semisolids should be added gradually and increased as indicated after the infant has shown its ability to first handle the milk mixture.

(b) Diets Composed Chiefly or Entirely of Starches. *Synonyms:* Flour injury, starch injury, mehlnahrschaden (Czerny-Keller).

Etiology. The condition follows feeding with a diet composed largely of cereals or cereal waters, as is frequently seen when these are used to replace milk mixtures which have been poorly digested (diarrhea, etc.). It is therefore due to continued feeding of flour gruels, either without milk or a diet too low in milk content. Whether simple flour or baby foods, dextrinized or not are used, the result is the same. Although the flour in its digestion is changed to sugar, the effects are not those of excessive sugar diet (acute), but only lead to acute symptoms after the organism has been generally impaired by the long use of the one-sided diet.

Pathogenesis and Metabolism. The disturbance of the organism which develops on one-sided flour feeding is to be regarded as qualitative inanition, being due to

the lack of important tissue-building substances (fat, proteins, salts and vitamines), and the resulting improper formation of the body tissues.

Steinitz and Weigert found in animals that a flour diet led to an abnormal chemical composition of the organism. The body became richer in water and fat than normal. The edema indicates a disturbance in the salt balance. Marriott believes that lack of fat-soluble vitamines is an important etiological factor.

In many cases, also, the caloric intake may be insufficient, so that quantitative inanition complicates the picture. The accumulation of large quantities of water which occurs when large quantities of flour are fed in presence of salts results in fluctuations in weight.

Rapidity of development depends on the following factors:

1. Age. The younger the child, the quicker the effects
2. The more the flour outweighs the other ingredients of the diet.

Symptoms. They may assume any form of nutritional disturbance. In many cases apparent symptoms of disease are lacking for a long time in spite of the improper diet. The infant may even apparently thrive well, since (due to the great water-binding property of carbohydrates) considerable gains in weight may occur. The appearance of the child is good, and fat cushion abundant. Even at this time, however, frequently some anomalies are observed: the musculature may be slightly hypertonic, the appearance may be pasty, suggesting a water-soaked sponge. Not infrequently by careful examination nervous irritability (latent tetany) may be detected. This is followed by development of grave symptoms of typical flour injury, which may assume variable appearance, according to whether the flour is given alone or combined with some other food.

Flour has the property of causing the body to take on weight by water absorption. This is especially true if

the infant was previously healthy, and may be misleading. In infants suffering from nutritional disturbances the picture develops more rapidly, especially upon inauguration of repeated starvation diet. Finally, however, both these groups of infants present the picture of an *inanition*—that is, the *atrophic stadium,* which cannot be distinguished from a *decomposition* clinically. They are subject to rapid weight and water losses, showing the loose binding of the water in the tissues.

Edema may complicate the picture, especially .where the flour is given in a salt-rich diet as bouillon, milk, etc., and the edema may resemble that of a nephritic patient (urine is usually negative).

The *natural immunity* in these hydremic conditions is greatly reduced, and the children are subject to furunculosis, otitis, and infections of the respiratory and digestive tracts, all of which give a bad prognosis.

Hypertonia is very common, with a characteristic muscular rigidity, resulting in stiffening of extremities, opisthotonos, etc., and it is often difficult to differentiate them from cases of spastic cerebral paralysis and chronic tetany, from which latter these infants often suffer. The history of nerve irritability must be used as a point of differentiation.

Hypertonic form has also been described, the chief symptom of which is the rigidity of the muscles. This hypertonicity may occasionally assume such proportions that the limbs and the entire body may become rigid. But this condition is not exclusively caused by flour injury, but may be seen also in other nutritional disturbances.

Stools. Often the stools are good for a long time, but sooner or later in all cases acute intestinal symptoms develop. More characteristic, after continued feeding on a one-sided flour diet are soft, mushy, loose stools, which are frequent, and vary in color from brown to yellow. A further characteristic is a tendency to fermentation, with

the formation of acids and gas, which tend to irritate the buttocks. The small, dark-brown stools, composed mainly of mucus (hunger stools), are not infrequently seen, and are of especial significance, because they are often misinterpreted as dyspeptic stools.

Diagnosis. The feeding history is of the utmost importance. Hypertonia and edema should lead to suspicion, as should the presence of excessive fermentation and "hunger stools."

Prognosis. The younger the infant and the longer the unsuitable diet has been continued, the worse is the prognosis. The high mortality in this condition is due not so much to the nutritional disturbance itself, but more so to unavoidably complicating infections. Tetanies and convulsions due to them are also grave complications.

Prophylaxis. The development of a primary flour injury is prevented by ordering proper diet. In using the flour diet for therapeutic purposes in the treatment of dyspepsia, especially when repeated starvation is inaugurated, the danger of development of the flour injury must be kept in mind, and the one-sided diet must not be continued longer than several days.

Treatment. 1. *Human Milk.* In young infants and also in all severe cases, feeding with human milk offers the best hope for the cure of the condition. It is absolutely indicated (1) before the third month, (2) in evidence of decomposition.

Begin with 200 to 300 mils daily, as in decomposition, and continue, even with weight loss and development of dyspeptic symptoms. Increase the amount steadily. Even with human milk the course will be slow, if the condition is well advanced.

2. *Artificial Feeding.* One-half boiled skim or whole milk plus water in feedings of 10 times 30 mils with water or tea *ad libitum.* Continue feeding, gradually increasing, unless the stools are dyspeptic. It is of advantage to hasten convalescence by addition of some sugar or mal-

tose-dextrin preparations to the milk mixture. Albumin milk and buttermilk mixtures are often taken to better advantage than whole milk mixtures. If they fail, human milk must be given. Codliver oil and orange juice should be started early in treatment.

If stools retain fat-soap character after 10 to 14 days, the diet may be more rapidly increased.

Course is often interrupted by weight drops and infections.

In very severe cases in which symptoms of athrepsia are present, same treatment as in athrepsia should be instituted.

(c) Diets Low in Vitamines. It is a well-known fact that even in the presence of diets containing proper amounts of fats, proteins, carbohydrates, mineral salts and water, retardation in the development and growth of the body may occur. This is frequently due to the lack of unidentified substances which are essential to life and which are described, for want of a better term, as vitamines. (See page 18.)

Beriberi, pellagra, scurvy and rickets may be wholly or partially dependent upon a lack of vitamines for their development. The so-called flour injury is also, in part at least, due to a lack of vitamines.

More recently it has been shown that many infants suffering from malnutrition and marasmus show a marked and rapid improvement in their general condition when foods rich in vitamines, more especially water-soluble B and C, are added to their diets in increased amounts.

Beriberi has positively been identified as a disease due to an insufficiency in water-soluble B vitamine.

Pellagra is in all probability a deficiency disease, although there is still considerable question, as to whether other factors may not be contributing.

Scurvy, judging from its prompt response to treatment with the water-soluble C vitamine, is undoubtedly a de-

ficiency disease, even though in many instances it does not become clinically evident until precipitated by secondary factors, such as an infection. (See page 359.)

Rickets. The pathological changes in the osseous and muscular system are undoubtedly due to the inability to utilize calcium, with a resulting diminished retention of this element, notwithstanding the fact that there may be a sufficient amount in the food intake and in the blood. Phosphorus probably plays an intermediate rôle in influencing the deposition of lime salts. It is quite probable that a fat-soluble vitamine is a factor in determining the level of the blood phosphate and thereby directly influences calcium retention.

Subacute and chronic infections, more especially of the respiratory tract, are seemingly influenced by administration of cod-liver oil, which is high in fat-soluble vitamine and which leads to the deduction that it has a more or less direct influence in raising the immunity through some unknown factor.

Treatment. The administration of diets properly constituted for the treatment of rickets is discussed on page 331 and scurvy on page 379.

In the presence of retarded growth fat-soluble A vitamine is best administered in the form of cod-liver oil, and water-soluble B and C vitamines, as contained in fresh fruit juices, more especially in oranges, and fresh vegetables, more particularly the green-leaf variety, are indicated.[1] [2] The infant's age permitting, orange juice should be administered in amounts equal to one to two ounces daily.[3] In a considerable number of this type of cases we have found the administration of one-half to one cake of fresh yeast daily of great value. It may be given in the orange juice or made into a paste with

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[1] Karr, W. G.: Jour. Biol. Chem., 44, 255, 1920; 44, 277, 1920.

[2] Osborne, J. B.: Medical Record, 97, 630, 1920.

[3] Byfield, A. E.: Daniels, A. L., and Loughlin, R.: Am. Jour. Dis. Children 19, 349, 1920.

butter or mixed with the fruit pulps, such as prunes or bananas. The paste may be spread on bread or crackers. When given in this way there is usually little objection to taste on the part of the infants. The same line of treatment is effective during the convalescence from acute infections and other debilitating diseases, and in the course of chronic infections.

CHAPTER IV.

NUTRITIONAL DISTURBANCES CHARACTERIZED BY DIARRHEA.
(Diarrheal Disturbances.)

THE following conditions, characterized by diarrhea as the most prominent symptom, are described in the literature: Acute intestinal indigestion, fermentative diarrhea, infectious diarrhea, dyspepsia, stadium dyspepticum, sugar indigestion (zuckernaehrschaden), fat indigestion (fettnaehrschaden), gastro-enteritis, summer complaint, cholera infantum, follicular-enteritis, membraneous-colitis.

Etiology. Diarrhea may develop either primarily in a healthy infant or a sequel to preceding nutritional disturbances or it may be secondary to an infection.

Diarrhea occurs as a symptom of many conditions of different etiology. Usually it is unassociated with demonstrable pathological lesions of the intestinal tract, and unfortunately these are the types most frequently seen. Anatomical lesions in the intestine, more especially in the lower ileum and large intestine, are constant findings in some of the infectious types and may be present in the subacute and chronic diarrheas, non-infectious in origin.

The most important factors may be enumerated as follows:

1. Overfeeding With:
    (a) Milk mixture of correct composition (too frequent or too much).
    (b) Milk mixture of incorrect composition (excess of fat, sugar or salt).
    (c) Raw milk, with resulting mechanical irritation due to large, hard protein curds.

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2. *Feeding with Spoiled Milk (decomposition products of milk and bacterial toxins).*

3. *Subnormal Food Tolerance Due to:*

   (a) Preceding dietetic errors and nutritional disturbances.

   (b) Extremes of temperature, heat of summer and cold of winter, with resulting systemic depression.

   (c) Constitutional anomalies:

      1. Idiosyncrasy to cow's milk.
      2. Exudative diathesis (eczema).
      3. Neuropathic diathesis.
      4. Organic diseases of the heart, kidneys, liver and pancreas.

4. *Infections:*

   1. Enteral.
   2. Parenteral.

5. *Cathartics (excessive and repeated administration).*

Very frequently several causes are combined in a single case, and it often becomes impossible to make an exact etiological diagnosis. In all varieties of diarrhea there are functional disturbances, and in the severe forms organic changes may be present, or the latter may develop in the course of an uncorrected case which at first was only functional in nature. An insufficiency of the intestines may soon be reached, which makes it impossible to avoid the development of pathological fermentation and leads to an interference with absorption of water and the products of digestion, with the early development of systemic derangement. The abnormal products of fermentation cause increased peristalsis and result in an aggravation of the condition unless corrected. Interference with the normal function of secretion leads to changed bacterial content in the intestines. It is, therefore, often impossible to determine whether this changed bacterial flora, as evidenced in the feces, is the cause or result of the condition.

Pathogenesis. The development of this group of disturbances will vary directly with the underlying causative factor or combination of causes.

Those Due to Overfeeding: With Milk Mixtures of Correct Composition. In this group vomiting and evidences of gastric and intestinal indigestion usually precede the appearance of diarrhea. In fact, many of the cases are unassociated with diarrhea, the infant presenting sufficient evidence of distress to call for reduction in diet before the latter stage is reached. The symptoms may be due to incomplete emptying of the stomach between feedings when the intervals between meals are too short, or they may follow overfeeding when too large or too concentrated a diet is fed. Sooner or later intestinal symptoms will develop unless the dietetic error is corrected, due to bacterial action on unabsorbed food in the intestinal tract.

In the second group due to *overfeeding with milk mixtures of incorrect composition,* any one or a combination of the food elements when in excess may precipitate a diarrheal disturbance. By far the largest number of cases as seen in practice fall within the limits of this group and we will discuss it more in detail because of the frequency with which it is met. The protein is not a good culture medium for the organisms commonly associated with the diarrheal diseases and, therefore, when fed in boiled mixtures, is not a common causative factor. On the contary, when casein is fed in sufficient amounts, in boiled, whole or skim milk or in dry forms, it counteracts pathological fermentation in the intestinal tract and the casein has a direct curative influence, as seen in the tendency to the formation of alkaline stools; on the other hand, when large quantities of raw milk are fed there is a tendency toward the formation of so-called hard protein curds which may act as mechanical irritants to the intestine, increasing peristalsis and secretion, with a resulting increase in number

of stools with a lessened consistency. This is rarely seen in the normal infant, unless more than one and one-half ounces of raw milk are fed per pound body weight. It may, however, develop on lesser quantities in infants who have suffered from previous nutritional disturbances. Far more frequent are the cases brought about by increased acid fermentation, which causes increased peristalsis and increased intestinal secretion with resulting loss of body fluids. Pathological breaking down of carbohydrates (sugar, flour) is the most frequent cause, and is often the primary factor, while the fat, in most cases, is involved only secondarily as a result of increased peristalsis and fermentation. The sugars in strong solution have a hydragogue action and they also provide a favorable medium for bacterial growth. While the fats may have a mechanical effect and thereby act as laxatives, when fed in excessive amounts this is only exceptionally the manner in which they cause diarrhea. Much more frequently, the irritation is due to the esters of the lower fatty acids which give rise to acids capable of increasing peristalsis when they are split by the enzymes of the digestive juices. This action is enhanced by an excessive carbohydrate fermentation. It is also true that an excess of fat has an unfavorable influence on the sugar tolerance. By the reduction or complete withdrawal of carbohydrates the pathological fermentation can in almost all cases be decreased and also the peristalsis. The different carbohydrates show different tendency to fermentation. Milk-sugar ferments most easily, less easily the cane-sugar, and least the maltose-dextrin preparations. By clinical experiments it was found that the tolerance of even the same intestine towards carbohydrates is not always the same, and that it also depends to a certain extent upon the quality of the fluid in which they are dissolved or suspended. The same amount of sugar given with large quantities of whey produces dyspeptic symptoms

much more easily than the same amount of sugar administered in less whey or in water. From this it follows that in pathogenesis of this group of artificially fed infants the whey is also of importance, the quality and quantity of the whey salts may become the deciding factor in the development of diarrhea. This group of cases is frequently described under the title of *fermentative diarrheas*. The *mechanical diarrhea* due to raw protein curds has been described—in older infants similar cases may be seen when the semi-solid foods are given, more particularly fruits and vegetables, among the latter those in which the vegetables are not properly puréed. Certain vegetables may also act as chemical causes, more commonly unripe fruit and green vegetables, such as apples, cabbage, cucumbers, etc.

Infected foods, such as spoiled milk, may result in acute disturbances which may be due to the bacteria themselves but more commonly to the bacterial action on the fats and sugars with the formation of toxic bodies. One of the greatest disadvantages of. feeding infants on commercially pasteurized milk, as is done in many of the large cities, is the fact that the non-pathogenic lactic acid organisms are destroyed and many of the pathogenic organisms remain which grow in the milk as it ages. Therefore, sweet milk does not necessarily mean good milk. The destruction of the lactic acid organisms prevents the souring of the milk which is of best aid in detecting stale milk. It is well known that milk may be sour and cause no symptoms. The action of such organisms as the *Bacillus lactis* and *Streptococcus lactis*, by the production of lactic acid, exert an inhibitory effect on many of the pathogenic organisms, while their own products are comparatively harmless.

Subnormal food tolerance due to *preceding dietetic errors* and nutritional disturbances, is one of the most important predisposing factors to recurrent diarrheal disturbances. Infants with such a tendency must be

kept under constant observation and the earliest evidence of a tendency to the development of a fresh attack must be given proper consideration so that the causative factor may be eliminated. These are the cases which so frequently pass into the stage of *athrepsia* and *anhydremic intoxication*.

Infections. Infants suffering from *parenteral infections,* such as tonsillitis, rhinitis, otitis, pneumonia and pyelitis, are likely to develop a diarrhea in the course of their infection. This is probably due to the fact that the infection lowers the functional capacity of the gastro-intestinal tract, results in lessened secretion of digestive juices, decreased absorption and an increased irritability. Usually the diarrhea is preceded by fever and often by vomiting. A careful study of the diet reveals no error in the feeding and there is usually a disproportion between the gastro-intestinal symptoms and the evidences of systemic involvement, more especially the fever. While this type calls for a reduction in the diet, prolonged starvation is to be avoided in order to prevent lessening of the infant's immunity. There is another probable explanation for the development of diarrhea in the presence of parenteral infection, in that in the presence of the general debilitating influence of these systemic infections the intestinal mucous membrane loses some of its anti-bacterial power and thus allows bacteria to flourish higher in the intestine than they would normally.

Enteral infections will be discussed in detail in a later chapter. (Page 284.)

Extremes of temperature, heat of summer and cold of winter, with resulting systemic depression, lead to digestive disturbances. It is a well-established fact that infants are greatly depressed by overheating of the body due to high external temperatures. The condition is readily aggravated by the wearing of excessive clothing during heat of summer; therefore, infants should be

dressed to meet the needs of the temperature of a given time in the day. It is wise to allow the mother to use her judgment as to over- or underdressing, after the possible effect on the child of overheating has been properly explained. Most infants will suffer more from heat in the presence of an excessive humidity and this should also be given proper consideration in considering the clothes for the infant's use. It is also true that less food is required during the summer months to nourish an infant, while at the same time more water is needed. These facts should be remembered and be taken advantage of as prophylactic and therapeutic measures. Unfortunately, this is not heeded in many cases, because the child is more thirsty, and, its food being liquid, quenches its thirst and is therefore given in excessive amounts; and secondly, because the cry and discomfort due to the same overfeeding and heat are interpreted as hunger. It should, therefore, be the duty of the physician to warn against excessive feeding during the hot summer months; that these latter are factors is evidenced by the fact of their prevalence among the poor and ignorant.

Certain *constitutional anomalies* lead to lessened food tolerance. Among these is an *idiosyncrasy to cow's milk* which is fortunately one of the rare disturbances. Usually the first feeding with cow's milk will result in acute vomiting, diarrhea and erythematous rash and not infrequently low grade fever. Withdrawal of cow's milk from the diet usually results in disappearance of the symptoms. Infants suffering from the *exudative diathesis* and infantile eczemas often show a tendency toward the development of diarrhea in the presence of minor dietetic errors, more especially so in the presence of parenteral infections, at which time there is a tendency on the part of the eczema to disappear which goes hand-in-hand with the increased irritability on the part of the gastro-intestinal tract. Some infants, from birth,

show a tendency to develop marked nervous manifestations upon slight external irritation. It is this class of cases which early develop *pylorospasms and repeated vomiting.* They are also subject to attacks of gastro-intestinal colic and they are often hypersensitive to light and sound. They form bad habits which lead to improper feeding due to misinterpretation of their cry. Intestinal disturbances are common complications. Even in the ordinary infant nervous exhaustion and excitement lead to impaired gastro-intestinal functioning and must be avoided.

Chronic diseases of the heart and kidneys, more especially when associated with decompensation, are frequently accompanied by diarrhea. These are in large part due to interference with elimination. Chronic disturbances of the liver and pancreas may be underlying factors in the development of diarrhea.

Repeated and excessive administration of *cathartics* is one of the most common causes of diarrhea. It has been conclusively shown that the stools become abnormal in the presence of repeated cathartics. Calomel, when given in divided doses of one-tenth grain per dose, with a total administration of one grain daily for three consecutive days, will cause the presence of·mucus and blood in practically every case; one dram of magnesium sulphate, given daily for three days, will cause the same result; castor oil, while less irritating, when repeated, will cause a similar reaction. It is self-evident that repeated catharsis is to be avoided.

Symptoms. Acute intestinal indigestion is characterized clinically by acute gastro-intestinal symptoms, the most marked of which are the stools, which are increased in number, and of an abnormal quality. In the milder types the organism does not show signs of any deep-seated general changes and weight loss is moderate. Quite commonly the gastric disturbances are associated with diarrhea, the causative influence being something

ingested which irritates the stomach and then passes into the bowels, or both may be affected simultaneously in the presence of systemic involvement. Temperature is moderately increased, and repair is rapid with the withdrawal of improper food. The presence of high or continued fever should lead to a careful search for a systemic involvement outside of the gastro-intestinal tract and for evidence of an indefinite infection.

Severe general symptoms are usually absent in the early stages. The mind is clear. The heart action is not rapid. Respirations are not greatly increased. The baby is restless and fretful, cries a great deal of the time, sleeps brokenly, and sucks its hands and other objects as if hungry. The face soon becomes drawn, and the tissues more or less flabby through loss of body fluids. The skin shows little change. The urine is diminished in amount, the quantity being dependent upon the amount ingested, and the extent of loss of fluids through vomiting and diarrhea and through loss by way of the skin. In the milder types there are no other abnormal urinary findings.

Weight. The weight loss varies directly with the loss of body fluids through the increased secretion, intestinal peristalsis and consequent diarrhea.

Gastro-intestinal Symptoms. The appetite is poor. The mucous membrane of the mouth is red, and may be the seat of thrush (due to decreased immunity). Vomiting may be present, and usually occurs long after feeding, more often preceding diarrhea by from twelve to twenty-four hours. Volatile fatty acids may be detected in the stomach content by their odor. The abdomen is distended, and peristalsis increased, and is visible or can be heard by auscultation. Restlessness is marked and is usually relieved temporarily upon passage of flatus.

Stools. The clinical diagnosis is usually made from the stools. They are increased in frequency, and they

also differ from the normal. They are thinner, contain more mucus, and are either watery or hashy. There is an abnormal odor, either that of decomposition or that of acid fermentation. The reaction is variable, mostly acid. The color of the stool is often green, this being due to transformation of bilirubin to biliverdin by oxidizing ferments. It is then passed without being reduced to urobilin, the normal transformation in the large intestine.

The increased peristalsis results in impairment of absorption, which may easily be determined by metabolic experiments, and also estimated by macroscopic, microscopic, and chemical examination of the stools.

Fatty acids and calcium and magnesium soaps appear in the stools in the shape of white or yellowish lumps, and, by addition of strong acids and slight warming, fatty acid needles may be crystallized from them.

Part of the fat is present in the form of smaller or larger neutral fat globules.

The acid reaction is in the greater part due to the fermentation of sugar which has escaped absorption in the small intestine and which is supplied by bacteria in the colon. When considerable amounts of sugar are fermented in this way the stools become foamy. The irritation of the colon also leads to excessive secretion of mucus and later blood may appear. The appearance of blood varies with the places of hemorrhage and the time which it is in contact with the intestinal contents. The presence of pus should lead to the suspicion that ulceration of the intestinal mucosa has taken place. The latter is rarely present in the simple types of intestinal indigestion and should be considered as a grave complication.

If flours are in excess, the stools are frequently paste-like and foamy. By iodine solutions the unchanged starches are stained blue, and the erythrodextrin is stained red.

Recent research has demonstrated the frequency with which casein is found in the stools. The yellowish lumps, the so-called milk-curds, in the hashy stools, seen even in feeding with boiled milk, have erroneously been regarded as casein curds. Today we know positively that these so-called "casein curds" are composed chiefly of fatty acid salts and bacteria. In feeding with raw milk large, tough, bean-like casein curds may pass through the intestine without being digested. Even in the presence of true casein curds, however, one must not conclude that they are the primary factors in the pathogenesis of this nutritional disturbance unless we are certain that an excess of raw milk has been fed.

Varieties. First, the *acute*, which begins with a definite acute onset, usually in infants who have been previously well, and second, the *chronic*, which begins less acutely, or follows acute attacks, and which recurs even in the presence of a carefully regulated diet. It soon becomes evident that in the latter cases there is a definite lessening of the food tolerance.

Diagnosis. The diagnosis can be made only by careful consideration of the feeding history and the clinical and functional symptoms.

It is first necessary to differentiate those unassociated with infection from the milder forms following enteral and parenteral infections. One must remember that the infections, especially in young infants, are frequently associated with a secondary nutritional disturbance, and *vice versa*, that secondary infections commonly follow in the wake of nutritional disturbances. An infection should be suspected when the temperature remains high after the withdrawal or reduction of the food (especially of the carbohydrates), and when albumin and hyaline casts appear in the urine, and the mucus continues in excess in the stools, presenting the picture of a secondary enterocolitis after the correction of dietetic errors. If infections are not recognized, there is a great danger

of continuing the starvation diet too long, and thereby reducing the vitality of the infant to the stage of athrepsia. It is also of importance to note whether it is a primary or an acute exacerbation in the course of an athrepsia, as on this differentiation to a great extent depends the prognosis and the therapy. Here, again, a careful history is of vast importance, and one should carefully note the presence of repeated attacks, with recurring fluctuations in weight, the occurrence of previous infection, both enteral and parenteral, as all of these indicate a tendency to malnutrition.

Prognosis. In infants previously healthy and with a proper dietetic treatment, the prognosis is good. Repeated attacks should always be seriously considered. Intestinal indigestion in very young infants is always more serious than in the older and better developed ones. The determination of the stage of nutritional disturbance gives us no indication as to whether we have a light or serious disturbance. What the physician needs to learn is that the condition of the infant should not be estimated only by its weight and the character of the stools, but also on the basis of its tonus and turgor, the color of the skin and the state of the sensorium. We must necessarily consider the nature and the quantity of the stools, since the diagnosis of this condition is in the first place determined by the presence of diarrhea. The degree of stomach involvement, however, must be based on the condition of the infant in whom it occurs. As a rule, most cases, if promptly treated, belong to the class of milder nutritional disturbances. However, these infants are always in danger of developing a general serious disturbance, more especially the very young and those who have suffered from repeated attacks as the result of long-continued starvation and excessive loss of water and body fluids, both of which tend to a destruction of the tissues. For practical purposes it is well to classify these infants, as to prognosis, into three types:

first, those who have been previously normal; second, those suffering from mild degrees of malnutrition, and third, those seriously emaciated either through constitutional defects or repeated nutritional disturbances. Among the latter group those that suffer from repeated diarrheal attacks are especially likely to succumb. A very acute nutritional disturbance prepares the field for the subsequent ones and decreases the tolerance for the various food mixtures. All diarrheal attacks have a tendency to result in the stage of malnutrition, and the severer ones in marasmus, which latter condition will be described in the chapter on Athrepsia.

The loss of water by way of the stools and skin and the diminished retention as a result of vomiting tend to a desiccation of the body tissues, which in the severe types leads to the characteristic clinical picture which will be described in the chapter on Anhydremic Intoxication.

Treatment. *Prophylactic Measures.* During the heat of summer and where there is any uncertainty as to the quality of the milk it should be boiled. It should also be remembered that less food and more water are required during the hot months. Over-clothing in summer and insufficient protection in winter predispose to diarrheal conditions. In the presence of diarrhea high carbohydrate feeding should be discontinued.

Human Milk. The best treatment of all forms of intestinal indigestion consists of feeding human milk. The younger the infant, the more the indication for human milk. This is especially true of infants under two months of age. In severe cases it may be necessary to place the infant on a starvation diet for six to twelve hours, and then administer the breast milk in restricted amounts.

Artificial Feeding. In artificial feeding the treatment of acute intestinal indigestion is somewhat different from the treatment of the chronic variety.

Acute Forms. In the acute form, where the child was previously well and-its tolerance good, the simple unloading of the intestine may allow it to resume its normal function. The following treatment is recommended:

1. *Starvation or Hunger Diet.* Short (six to twelve hours, rarely longer) starvation, only liquids being administered, tea with saccharin being the best (saccharin, 1 grain (0.065 Gm.) to 1 quart (1000 mils)). They should be given freely, up to amounts of the total fluids needed. This permits the stomach and the intestines to empty themselves, and to assume their normal functions. Laxatives are usually not indicated. If temporary starvation is inaugurated, the intestinal tract soon empties itself of its irritating contents.

Upon suspicion that spoiled or otherwise improper food has been fed, a single dose of cathartic may be indicated. Of these the least harmful are castor oil and milk of magnesia. The repeated administration of laxative drugs is absolutely contraindicated. This applies more especially to calomel.

2. *Indifferent Diet.* During the second day in young infants, one-third whole or skim milk (boiled five minutes) plus two-thirds water or thin oatmeal gruel, without sugar, may be fed, such a diet being low in food value and salts. The total daily quantity of the milk mixture on the second day should not exceed six to eighteen ounces (180 to 540 mils), divided into six feedings of one to three ounces each. To this, twenty to twenty-five ounces of tea, plus saccharin, may be added, making a total of at least one quart of fluid for the day. Even better results will be obtained by the use of lactic acid milk, at first fat-free and later whole. These can be prepared by inoculating boiled milk with a pure live culture of one of the lactic acid-producing organisms which can be obtained on the market. The preparation of the lactic acid milk can be begun with the institution of the starvation period, as it must be inoculated from

twelve to twenty-four hours, after which it must be kept on ice. The lactic acid milk can be fed in somewhat larger amounts than sweet milk. This is probably due to the fact that the lactic acid and bacilli has a retarding action on the growth of many of the other organisms which may be present in the intestinal tract. Such an action is especially valuable when it affects the growth of the abnormal flora in the upper intestinal tract. Knowing that the irritation, more particularly of the small intestine, interferes with the process of digestion and absorption which are so necessary to the relief of this class of infants. In young infants and in the severer cases lactic acid milk prepared from skim milk is to be recommended over that made from whole milk during the first stage of the treatment.

Further treatment depends on the reaction to the above. Upon this treatment the general condition improves, also the disposition, etc., and the weight loss ceases in two or three days. When this is not the case, infection should be suspected.

3. *Sustaining Diet.* Gradually, and as rapidly as possible, the food should be increased, the increase to be made at least every other day, in order to limit the underfeeding to a minimum. By the third day or before, the quantity of food should be increased, the quality may be left unchanged, giving water or tea to the necessary quantity of fluids between the feedings. Weight increase should not be expected because of the low sugar content and low caloric value of the diet, but a decrease in weight should always be considered serious. The stools are at first small and contain mucus, later less frequent, and often on milk mixtures without sugar fat-soap stools soon appear, which is a good indication.

4. *Ordinary Diet.* In mild cases, the ordinary milk mixtures proper for the given infant may usually be resumed by the end of a week. In more severe cases, return to a full diet should be slower. In these mix-

tures, the carbohydrates should be started by adding one gram and gradually increased to four or five grams for each pound of body weight, only exceptionally, however, should the total addition of sugar exceed forty-five grams (1½ ounces). The carbohydrates most suitable for this purpose are the maltose-dextrin compounds, especially those with a high dextrin content and no potassium carbonate. Corn syrup is also valuable, being in part at least absorbed in the upper intestinal tract and thereby causing less intestinal irritation. It may be added in amounts of 15 to 60 mils to the day's total food. In older infants cereals, in the form of flour ball. barley flour, farina, zweibach, can often be added to advantage, as well as clear broths. At first there is a rapid increase in weight, later on a slower one.

Avoid underfeeding too long, even if the stools look bad, if the temperature and weight curves improve, because of the danger of athrepsia. It should be borne in mind, therefore, that it is undesirable to underfeed for a long period, and more especially dangerous to inaugurate starvation repeatedly, or to keep an infant for days on a starvation diet, such as cereal waters or very weak milk mixtures. It is also necessary to know and recognize the stools of an underfed infant *(hunger stool)*. This is greenish-brown in color, composed chiefly of mucus, and small in amount, and sometimes frequent. They should not be mistaken for the curd-containing, frequent stools of intestinal indigestion, as the former is an indication for the resumption of food, while the latter indicates starvation. Fats can be added in place of sugars, but this should be done with care. Cod-liver oil has given us the best results. It should be given in small quantities at first, beginning with one mil twice daily, and increased to four mils per dose.

In some infants the above-described treatment is unsuccessful. In one group of these cases the loss of weight is not favorably influenced, while the stools im-

prove; and in a second group the loss continues with continued diarrhea. In these cases there is either infection or they are cases of grave nutritional disturbances on transition to athrepsia. It would be a very great mistake to continue starvation longer, with the idea that by giving the digestive tract longer rest, it may still recover. This may kill the child. In these cases treatment as recommended for athrepsia or infection must be instituted. Therefore, it is advisable to use routine treatment as described above, and, if not successful, the underfeeding should not be continued under any circumstances, but the treatment for athrepsia (described later) or infection (see Infections) should at once be instituted, if human milk is not obtainable.

It is in these cases that Finkelstein's albumin (protein) milk is indicated. (See Appendix, for preparation.) The albumin milk may be administered undiluted in the same quantities recommended for the lactic acid milk and increased three ounces (ninety mils) daily until three ounces per pound (180 mils per kilogram) are administered.

The value of feeding with the lactic acid milk and albumin milk is due to the presence of the lactic acid bacillus and its product, lactic acid, and their high protein and low sugar content and their small curd. The latter also has the added advantage of having a low salt content. After the stools become firm, sugar should be added to both the lactic acid milk and the albumin milk, beginning with one-half to 1 gram and gradually increasing to four grams for each pound of body weight, with a maximum addition of forty-five to sixty grams. Carbohydrate starvation, more especially in the presence of high fever, is an added danger, and addition of sugar to the diet may be imperative, even in the presence of soft stools. Maltose-dextrin compounds which do not contain potassium carbonate are the best for this purpose. We have found that 1 per cent. of flour (flour ball)

may be added to the albumin milk, thereby raising its caloric value without decreasing its efficacy, at the first feeding. After three or four weeks, in both mixtures, it is usually safe to replace them in part or entirely by a suitable milk formula for a child of a given age. It is usually wise to reduce the sugar in the formula during the first days of the new feeding.

When it is not practical to prepare the lactic acid milk or the albumin milk in the home, the albumin milk may be used in the dry form, in which it can be obtained on the market (See Appendix).

The protein content of the sweet milk mixtures can also be increased when desired by the addition of one of the casein products which are now to be obtained in quantities varying from 1 to 3 per cent. of the milk content in the mixture (See Appendix).

For the treatment of the severe types with toxic symptoms, see Anhydremia. The treatment of enteral infections is discussed in the chapter on Infection and Nutrition.

Chronic Cases. In treatment of chronic forms there is no indication for underfeeding. Since here there is no transitory weakness, but a chronic weakness of tolerance, the additional trauma of starvation would have an unfavorable influence. Fats must be reduced. Skim milk, buttermilk and albumin milk are often better taken than whole milk mixtures. Carbohydrates are to be reduced to the infant's minimal needs (two to four grams per pound body weight), and the less easily assimilable carbohydrates may be replaced by those that are more easily assimilated (maltose-dextrin mixtures or corn syrup). If this does not improve the stools, then nursing on the breast is necessary. The quantities of foods taken should be carefully measured and recorded to prevent prolonged underfeeding with the hope that when the child becomes older the tolerance will become physio-

logically increased, and the condition thereby undergo spontaneous healing.

Medicinal Treatment. This is unnecessary in most cases. When the starvation period shows no tendency to decrease the number of stools and where there is considerable pain and flatulence, it may be necessary to administer small doses of opium and atropin. Paregoric is the safest form for administration of opium. Depending upon the age, it may be administered in doses from two to ten minims, to be repeated as indicated by the effect on the gastro-intestinal tract and the general symptoms. If atropin is used, the doses must be carefully graduated, in young infants, and should range from $\frac{1}{1500}$ to $\frac{1}{500}$ of a grain. Epinephrin in $\frac{1}{1000}$ solution, 1 to 5 minims by mouth, may be indicated where there is marked atony on the part of the intestinal tract. A pure culture of lactic acid, when feeding sweet milk mixtures, is often of value. It may be administered in the form of powder or liquid culture.

Marriott has recently recommended the administration of argyrol. He advises 6 grain doses prescribing it in a 10 per cent. solution, of which 4 mils are given in each feeding. It can be continued for from two days to two weeks. He believes that this has an inhibitive action on the growth of the bacterial flora in the upper intestinal tract. The stools are stained a deep brown when such medication is used.

For the treatment of irritative conditions which persist even after the intestinal indigestion proper has disappeared (loose stools in presence of gain in weight), astringents are of use. Tannigen or tannalbin, 1 to 5 grains (0.065 to 0.325 Gm.) four to five times daily, will answer, or calcium lactate in doses of 10 to 15 grains (0.65 to 1 Gm.) may be prescribed in a 10 per cent, solution to be added to each milk feeding.

CHAPTER V.

ATHREPSIA (DECOMPOSITION).

Synonyms: Malnutrition, marasmus, infantile atrophy, pedatrophy.

The term athrepsia was first used by Parrott in 1877 to describe secondary nutritional disturbance with resulting severe malnutrition. The same symptom-complex is described as the "stage of decomposition" by Finkel-
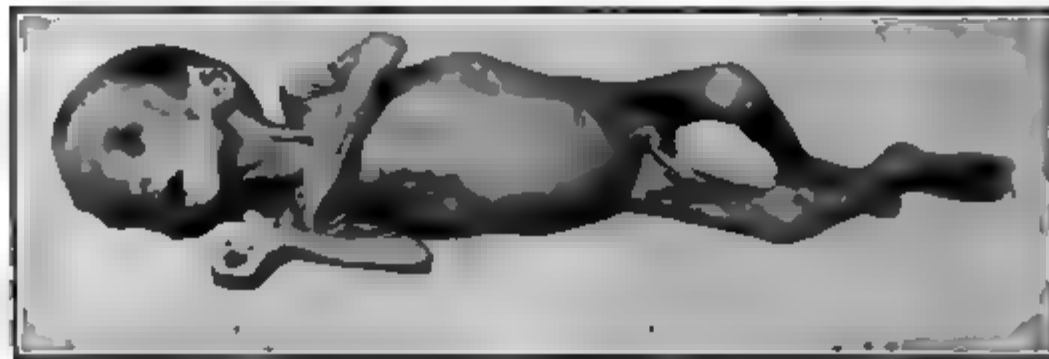
Fig. 9.—Infant with athrepsia (decomposition).

stein. The milder types of athrepsia are, in the American literature, described as cases of malnutrition and the more extreme grades as marasmus. The cases will range in severity from those which simply show an insufficient gain or a stationary weight with few systemic changes, to the most extreme types, which will be described more in detail.

The clinical picture may be viewed as the end-result of repeated nutritional disturbances or constitutional factors. The result of such prolonged or repeated undernourishment must necessarily be malnutrition, of a more or less marked degree, depending upon the disproportion between the food utilized and the needs of the body for energy and growth.

The past history of the patient is of the utmost importance, and a careful search reveals improper diets,

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with resulting disturbance of nutrition, or a nutritional disturbance following enteral or parenteral infections, each leaving in its wake evidence of impaired nutrition, until after weeks or months we have reached the stage of deep-seated tissue starvation. Most frequently athrepsia results from repeated injuries and therefore is classed as a subacute or chronic condition. It may, however, develop more rapidly in the presence of an acute diarrheal disturbance. The chronic infections, such as syphilis and tuberculosis, pyelitis and otitis, may also result in a similar picture, but must be differentiated to clear the classification for therapeutic purposes. It is very commonly seen in infants suffering from congenital weakness or disease, such as prematurely born infants and those suffering from congenital heart, pulmonary, gastro-intestinal and cerebral lesions.

During this stage it becomes increasingly difficult for the infant to assimilate a sustaining diet, with resulting extreme loss of weight, and of resistance of the organism to infections and other injurious external influences (heat, cold), this general weakening of the vitality of the infant being due to perverted metabolism, consisting of breaking down of the body substance, and change in the composition of the cells (abnormal katabolism), and of deficient and improper assimilation of the food (abnormal anabolism).

Etiology. The milder types of malnutrition due to dietetic errors are usually direct forerunners of the stage of athrepsia. All the factors which lead to diarrhea disturbances, anhydremia and intoxication may also be causative factors of athrepsia. At what moment this change takes place we have no means of telling, but we know that deep-seated organic changes are necessary to its development; these changes which produce such an intolerance toward nourishment may have developed previously to the preceding illness, or during its course. Premature infants are especially predisposed, also young

infants with previous dietetic errors and diarrheal attacks, also those fed on a one-sided diet, excessive in carbohydrates, especially cereal waters and gruels, as seen in too long continued "starvation diet." Especially to the very young does the statement as to cereal waters and gruels apply. All of the preceding reduce the tolerance toward assimilation of a full and normal diet. The tendency to athrepsia, and therefore to the narrowing of the nutritional sphere increases with each diarrheal attack. Czerny's internal hunger, or, as he commonly calls it, "cell hunger," is the cause of athrepsia. The above term is used in contradistinction to hunger as usually thought of, which is due to a lack of food to appease the appetite.

While a considerable number of the cases of athrepsia are due primarily to underfeeding, such as by nursing on dry breasts or in artificial feeding by the giving of food mixtures which are insufficient to meet the infant's needs, either because of a too small quantity or prolonged feeding of an improperly balanced diet which is too low in some of the necessary elements, the majority of the cases follow in the wake of the acute nutritional upsets which may or may not be based upon previous errors in diagnosis. Following such digestive disturbances the time is soon reached when even in the presence of a feeding of sufficient calories which are properly distributed, the functions have become so impaired that the products of metabolism cannot be utilized by the body tissues.

Pathogenesis. In the American literature the term marasmus is quite generally used to describe the clinical picture as presented by the severe types. It was assumed that the destructive changes in the intestinal glands following chronic inflammation, with a secondary impairment of the functions of absorption and excretion, were the underlying pathological conditions, which resulted in an inanition. It is, however, rarely possible to demon-

strate pathologic involvement of the secretory glands, even in the severe types. Marriott has suggested that due to the fact that there is an atrophied and poorly circulating blood stream, it must necessarily result in inefficient functioning of the digestive glands and a diminished absorption from the intestinal tract, both of which would necessarily be important factors in the development of the clinical picture.

Every organ of the body suffers more or less from the effects of an insufficient food supply. The blood volume is markedly diminished as well as the volume flow, so that the amount of blood passing through a given part of the body in a unit of time is much diminished under the normal. Marriott and Perkins[1] found that in normal infants under one year the average blood volume was 9.1 per cent. of the body weight. In a group of eleven athreptic infants the average was 8 per cent. of the body weight, the most extreme variation being 4.8 per cent. It should be remembered in considering the figures for athreptic infants that these infants were greatly emaciated and under weight and that, therefore, their blood volume was reduced to a greater extent than the percentage figures would at first lead one to believe, showing that there had been a very considerable destruction of the blood coincident with the loss of other body tissues. Along with these figures in blood volume they also found the volume flow to be decreased often to less than one-fifth and occasionally to less than one-tenth of the normal. During convalescence the volume flow increases with the treatment. They also found a concentration of the protein in the serum of these infants, also a diminution in the red blood cells and hemoglobin. This may be further aggravated by myocardial weakness as the heart muscle undergoes the same destructive changes as do the tis-

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[1] Marriott, W. McKim: Amer. Jour. Dis. of Children, xx, 461, 1920.

sues in general. The blood pressure sometimes falls below normal, due to the fact that in addition to the other pathological conditions in the circulatory system there is an absence of increased viscosity of the blood. The decreased blood volume leads to a constriction of the arterioles and this leads to a piling up of the corpuscles in the capillary blood. The adipose tissue disappears and there is an autophagia of the protein elements and a chronic loss of water and of mineral substances. The earlier stage may be described as one of the hypo-athrepsia and the late severe types as that of athrepsia. In the breast-fed the severe types are rarely seen unless preceded by intercurrent disease. In the artificially fed the stage of hypo-athrepsia may progress to athrepsia even after the primary digestive disturbances have subsided and in the presence of insufficient or improper diet. This condition is especially prone to develop in the environment of institutions and asylums unless the nursing care includes a sufficient amount of "mothering" and massaging of the infants. The supervision of the feeding of infants is of extreme importance, so that they may be sure of obtaining their diet in its entirety and while it is warm.

The great and sudden fluctuations in weight, as seen in this condition, must in the first place be due to loss of water and salts, while the disintegration of the body substance, other than the blood, including the cells, furnishes only a smaller proportion of the loss of weight which occurs.

Further, the abnormal splitting of sugar and fats contained in the food produces excessive amounts of acids in the intestines, which results in the loss of alkali salts, first, through neutralization of the acids formed in the intestinal tract from the food, and secondly, through salt losses due to excessive intestinal secretion, due to irritation of the bowel. As a result of such enteral loss of salts an increased NH-excretion takes place, which

is evidenced clinically by increase of ammonia in the urine.

To cover these losses, salts deposited in the tissues are in part withdrawn, and finally the cells themselves are destroyed through being deprived of their salt content (mineral hunger). It should be remembered that an abnormal fat metabolism is frequently the essential factor in the etiology of this condition, due to an overstepping of the fat tolerance. And further, that fermentative changes in the carbohydrates produce increased acidity of the contents of the intestinal canal, and so enhance the action of fats. Both of these may be causative factors in the development of diarrhea. While there is usually an excess of protein loss over protein assimilation, the tolerance for proteins is usually less affected. Because of the loss of nitrogenous substances, due to a relative excess in excretion of NH, proteins must be utilized in the diet to counteract these losses.

There is no increase in the osmotic pressure of the blood and hence no diminution in the amount of urine secreted and no accumulation of the urinary waste products of the body. Such acidosis as may occur is to be ascribed to causes other than retention of the acid products by the kidney. The *acidosis* is probably in part due to the diminished volume flow of the blood through the tissues and to a lesser extent to the production of acetone bodies, the result of partial starvation. Acidosis is, however, by no means a prominent feature of this condition and rarely calls for alkali therapy.

Increased peristalsis in diarrheal conditions results in further inanition, due to the passing of undigested food through the intestinal tract. All of this results in decreased assimilation of food necessitating the burning of the infant's own body for fuel. The stored carbohydrates (glycogen) and fat are first used and later also the body protein is consumed. When this latter stage

is reached the maintenance of life for any considerable time is impossible.

In these infants in the presence of impaired function on the part of the digestive tract it is quite possible that there may be an invasion of the upper intestinal tract by a bacterial flora which, under normal conditions, does not thrive in this region. This may act in two ways, by interference with the normal processes of digestion and absorption in the region involved, and by the setting up of inflammatory processes which may result in the involvement of the entire tract with the development of diarrhea.

Marriott[1] believes that the comparative buffer value of the food which the infant is receiving may be a factor in the development of malnutrition in some infants. By buffer value is meant the capacity to unite relatively large amounts of acid or alkali without a change in chemical reaction. If the same amount of hydrochloric acid is added to equal volumes of human milk and cow's milk it is found that the acidity of the human milk, expressed in terms of hydrogen ion concentration, is far greater. When milk enters the stomach of a normal breast-fed infant gastric juice is secreted in such amounts that the stomach contents ultimately reach a certain degree of acidity. This acidity, expressed in terms of H-ion concentration, averages about 1×10^{-5}, which is the optimum concentration for rennin action and is sufficient to markedly inhibit bacterial growth (Hahn[2]). Suppose, however, cow's milk, instead of breast milk, is fed: If the same amount of gastric juice is secreted, it is entirely insufficient to render the stomach contents acid to anywhere near the same optimum degree. To bring cow's milk to the optimum acidity of 1×10^{-5}, at least three times as much hydrochloric acid is required as in

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[1] Marriott, W. McKim: Amer. Jour. Dis. of Children, xx, 461, 1920.

[2] Hahn: Am. Jour. Dis. of Children, vii, 305, 1914.

the case of human milk. (This may readily be demonstrated by titrating breast milk and cow's milk with diluted hydrochloric acid, using as an indicator neutral red which changes color at a hydrogen ion concentration of about $1 \times 10{—}^5$.)

It is interesting to note that those foods which have been found empirically to be the best tolerated by athreptic infants are those which have a low buffer value or in which the buffer is already partly neutralized by acid. Breast milk, well diluted cow's milk with added carbohydrate, lactic acid milk and protein milk are examples.

Symptoms. The cardinal symptoms of athrepsia as concern nutrition are the inability to utilize food and the negative nitrogen and mineral salt balance. The clinical picture presented is that of a wasted infant, which may be of any degree of severity. It is often impossible to interpret the beginning of this severe form of nutritional disturbance. As a rule there is a history of repeated minor disturbances with a gradual impairment of metabolism. In other cases it may develop more rapidly and this is especially true in the presence of diarrheal attacks. Characteristic of the severer types is the development of diarrhea upon increased feeding and continued weight loss when the diet is insufficient to meet the infant's needs. Often it is preceded by an acute parenteral infection or the lighting up of a chronic infection, such as otitis, pyelitis, or bronchitis. The advanced cases present the following clinical picture:

1. Lack of ability to assimilate food is pathognomonic of this condition. The paradoxical reaction to food, mentioned in the two preceding stages of nutritional disturbances, becomes here a striking and serious phenomenon. Starvation or the institution of the *hunger day* as a therapeutic measure in these infants not infrequently results in an inanition which is fatal to the infant. Again, too rapid increases in the diet are equally serious, and not infrequently precipitate alarming and
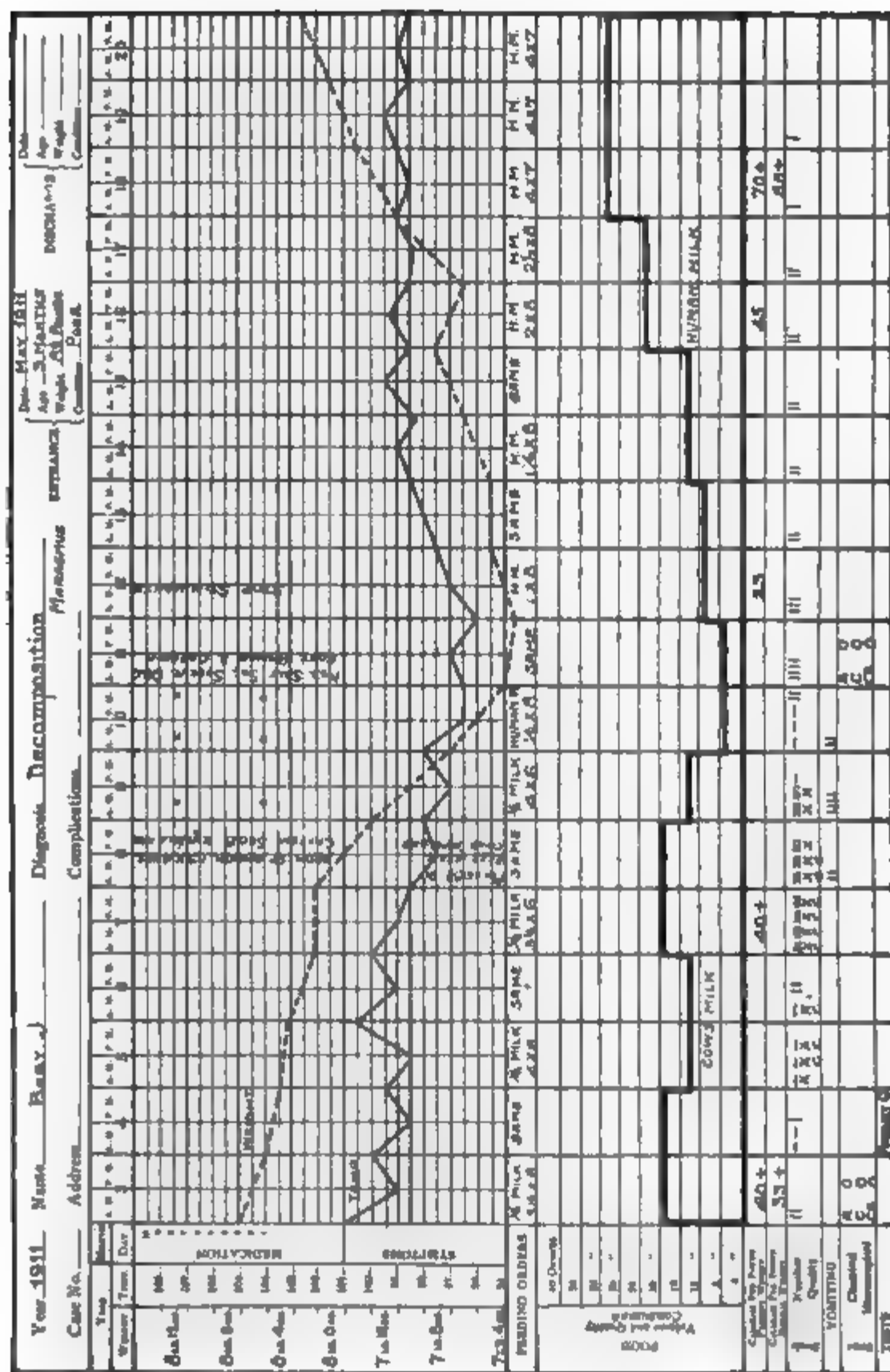
Fig. 10.—Chart showing hospital record of infant with athrepsia (marasmus, decomposition).

fatal symptoms. When the condition has progressed to this degree, human milk alone offers hope of recovery.

2. Loss in weight is the second cardinal symptom of athrepsia, due, as the name of this condition suggests, to disintegration of the body substance. This may be slight in the beginning, and in the light cases; in the later stages and in severer cases, however, it is often sudden and rapid, and may reach daily losses from 1 to 3 ounces (30 to 100 Gm.), resulting eventually in a picture of marasmus. The baby becomes thin, emaciated, wrinkled, with prominent ribs, covered with tightly drawn skin, and with intercostal spaces deeply marked (skeleton-like). The tissues are soft and flabby, the muscles either relaxed or hypertonic, the abdomen protuberant, usually distended; the color, pale first, later changing to characteristic grayish-white, with more or less cyanotic lips, fingers and toes. The mouth appears large, the cheeks sunken, and the facial expression anxious and serious. These characteristics give to the infant the appearance of a wrinkled, old man. As has been previously stated, in the earlier stages, these babies are irritable and apparently in constant distress, cry a great deal, and are excessively hungry. In the later stages, however, they are often apathetic, and apparently too weak to perform voluntary movements. When they have reached this stage, they are subject to sinking spells—that is, periods in which their vitality is very low. These may become very alarming, and often result fatally.

3. Vomiting is frequent.

4. The hunger is often very great, and extremely difficult to satisfy.

5. Subnormal temperatures, ranging from 96° to 98° F., with an irregular daily curve, is the rule. The temperature can easily be raised to 100° F. or more by the application of artificial heat (hot-water bottles, etc.), and

can sink quite as rapidly and alarmingly when the artificial heat is removed.

6. The pulse is often slow and small, and the heart-beats weak, and often only one heart tone is heard at the apex. The blood is thin, pale, and has a low hemoglobin and red cell count. There may be a moderate leucocytosis. Collapse is likely to result from circulatory failure, suddenly and without warning.

7. Respiration becomes rapid, and the expirations prolonged. The breathing becomes irregular, even to the Cheyne-Stokes type.

8. The sensorium is not involved in these infants, and when not too weak they take cognizance of their surroundings, are alert, and sleep but little.

9. The urine usually shows an increased ammonia coefficient. It may contain albumin, but very rarely sugar.

10. The stools are variable, mostly dyspeptic, occasionally diarrheal. In the earlier stages and in periods of remissions they may be quite firm (soap stools), again soft and firm stools may alternate. The hunger stool—small, dark, and containing much mucus—is common, especially in advanced cases, with an inability to take proper diet. Dark-brown, black, and tarry stools indicate, usually, hemorrhages from ulcers in duodenum (Helmholtz). We therefore learn to recognize the character of the stools as being only of secondary importance in the diagnosis, and also of secondary importance for treatment. We must not be misled into further starvation because of temporary changes in character, even for the worse, of the stool, due to the changes in the diet instituted for therapeutic purposes.

11. These infants are peculiarly susceptible to infections, and even slight infections of the skin, respiratory, gastro-intestinal, and genito-urinary tracts, may prove fatal.

12. Edema, cyanosis, and a more or less generalized purpura are not infrequently forerunners of an impend-

ing death. The development of edema, with corresponding gain in weight, may lead to the conclusion that the infant is improving. During this stage there is always great danger of the development of diarrheal complications.

13. Acidosis may develop and is probably in part due to the diminished volume flow of blood through the tissues, and to a lesser extent to the development of acetone bodies, the latter due in part to starvation. As there is usually no diminution in the amount of urine secreted when sufficient fluids are administered, the acidosis is not due to an accumulation of urinary waste products in the body. Acidosis is usually not a prominent feature.[1]

Diagnosis. The diagnosis in severe cases may be made from the clinical picture of the condition, but it is necessary to exclude emaciation due to tuberculosis, syphilis and cachexia caused by other disease, and also by simple inanition, due to prolonged underfeeding. This is to be based on the history and examination of the infant. In lighter cases it is necessary to differentiate especially from disturbed metabolic balance and from simple dyspepsia, since the treatment which improves these conditions may do considerable harm in infants suffering from athrepsia. The status praesens is not sufficient for making the diagnosis, since, as previously mentioned, remissions with stationary weight and good stools often occur. In these cases the history is of utmost importance: repeated diarrhea, loss in weight and febrile infections should lead one to suspect athrepsia. The positive diagnosis is made upon the reaction of the infant to food. If on somewhat increasing the diet a marked and severe paradoxical reaction appears (diarrhea, loss of weight, and occasionally fever), athrepsia should be suspected.

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[1] Marriott, W. McKim: Am. Jour. Dis. of Children, xx, 461, 1920.

Prognosis. We must remember that while primarily the picture of the disease is a nutritional one, the death is frequently brought about by infection. The younger the infant the greater is the mortality and this is most especially true during the first months of life, when breast milk is not obtainable. Among older infants the prognosis is better. The hygienic conditions under which the infant is treated and the care with which the treatment is carried out are important factors. On the whole, these infants do better in the home than in the general wards of institutions, due to the fact that they require a great deal of individual nursing care. In institutions there is also the possibility of infection through exposure. Convalescence is usually slow and may cover a period of several months in the severe types.

The prognosis depends on the following factors: (1) The stage of athrepsia. When the loss of weight has reached one-third of the body weight (Quest's figures), then the reparation, under any treatment, seems to be impossible. (2) The nature of the dietetic treatment, and especially the possibility of feeding with human milk. If one avoids the common errors, even the severe cases may be saved, except when the treatment is started too late. (3) The extent of the lowered immunity. The prognosis should always be guarded.

Improvement is common, even in severe cases, but there is a great tendency to sinking spells and collapse. Death in these cases is sometimes remarkably sudden. It usually occurs in one of the following ways: (1) By sudden syncope. (2) By apparent paralysis of the respiratory center. Periods of apnea usually precede the latter. There is no disturbance of consciousness. The face looks gray, and the eyes are staring. The breathing becomes irregular and slow, the heart weakens, the temperature sinks far below normal. Cyanosis increases, and breathing gradually stops. Sometimes the heart stops first. Such death may extend over days.

Treatment. Prophylaxis is the key-word to success-
ful treatment. A recognition and proper interpretation
of minor nutritional disturbances will avoid the graver
conditions.

For a proper conception of the therapeutic needs we
must recognize:

1. That we have a chronic condition which is subject
to acute catastrophes.

2. That the younger the infant and the greater the
preceding dietetic errors, the graver are the consequences
of athrepsia.

3. That starvation is dangerous.

4. That food is assimilated with difficulty.

5. That the downward weight-curve is likely to drop
suddenly with improper feeding and intercurrent infec-
tion.

Three essentials are necessary to the successful treat-
ment of the majority of cases of athrepsia: (1) Avoid-
ance of prolonged starvation; (2) the administration of
sufficient inert fluids, and (3) human milk. It is the
misfortune of most of these infants to have their ab-
normal stools—or more commonly, the hunger stools
previously described—interpreted as an indication for
starvation, regardless of the fact that the baby is already
starving. It has been our experience not only to have
seen one day of starvation, but repeated periods of
starvation, the rule, because of misinterpretation of the
significance of the "starvation stools." A single day of
starvation is often sufficient to kill an advanced case,
and even prolonged underfeeding, below 60 calories per
kilogram (the amount required to sustain the body equi-
librium), has a very harmful effect. Starvation from
without is thus added to inanition from within.

1. *Water Administration.* Athreptic infants must at
all times receive sufficient fluids to meet their needs.
They should, during the twenty-four hours, be given
approximately one-fifth of their body weight in water,

including that contained in the food administered. The water or weak tea feeding should be set aside in a sterilized bottle and given between feedings.

2. *Feeding With Human Milk.* It must be given in moderate quantity, best guarded by drawing off and feeding, as these infants drink too rapidly (always hungry), and do not stand large amounts. About 200 to 300 mils daily is enough to sustain the infants temporarily (70 calories per kilogram is sustaining—Rosenstern). Feed often; ten feedings may be given, one every two hours (20 to 30 mils), weak tea or saccharin water *ad libitum* between feedings. The daily quantity should be increased as rapidly as possible (at least every other day), until not later than after seven to ten days about 100 calories (130 to 150 mils) per kilogram are administered. The number of feedings should gradually be decreased as the condition improves, and direct nursing on the breast may be tried later, but the danger of overfeeding must not be overlooked. There is usually little danger of overfeeding when there are no gastro-intestinal symptoms and the diet consists exclusively of breast milk. Because of the decreased weight little can be expected in the way of rapid progress until the diet reaches a quantity which would approximate 100 calories per kilogram for the average full-weight infant at a given age, which not infrequently means 150 to 200 calories per kilogram for the athreptic infant. The diet should be steadily increased as long as there are no gastro-intestinal disturbances.

Weight may still not improve for some time. This Keller calls *"reparation stage."* Even on feeding with human milk there is a shorter or a longer period of stationary weight (depending on the severity of the case), which, however, is accompanied by improvement of the symptoms. Those who have not had experience in these cases may be inclined to blame the wet-nurse, and advise a change. In the stage of reparation, how-

ever, the body is being reconstructed, without being able to put on weight, this being partially, at least, due to still deficient absorption, and partially also to the fact that the human milk, containing comparatively small quantities of proteins and salts, furnishes only a limited quantity of material for rebuilding of the body. Only after this period the gain in weight begins and it may be shortened by feeding daily 100 mils of boiled buttermilk or skim milk, which is rich in salts and proteins, both of these substances hastening weight increase. This is not to be done until after the second or third week of treatment, and with a close observation of the results. It may be fed by mixing with and distributing through the expressed breast milk.

It may be necessary to add carbohydrates to the breast milk, more especially when the quantity at hand is insufficient. This should be done with considerable care, the daily quantity added being increased by four to eight grams at a time. This may be given in the form of cane and milk sugar or corn syrup. In older infants the cereal gruels may be added to the diet during the reparation stage.

The complete recovery is not to be expected sooner than in two to three months. And only then should the return to artificial feeding be thought of. The weaning should be preceded by experimental administration of a small quantity of whole cow's milk, as there is a possibility of idiosyncrasy to cow's milk.

3. *Artificial Feeding.* If there is no possibility of feeding an infant suffering from athrepsia otherwise than with artificial food mixtures, then much the same rules are to be followed as have been given for diarrheal disturbances. As has been previously stated, there is a great need for food and there always exists the danger of overfeeding, which holds particularly true in those who must be put upon artificial diet. We must, therefore, often be satisfied with a very slow stage of

reparation and expect little gain in weight until the infant is able to assimilate sufficient quantities of food, which may mean a diet with food value considerable over that required by the normal infant. Our best results have been obtained by the administration of whole and skim lactic acid milk and albumin milk (protein). The two latter foods, however, have a very low caloric value, which must be increased by the addition of sugar and starch.

It may be necessary to use boiled whole or fresh skim milk when the lactic acid milk or albumin milk is not available. The feeding with these foods will be described in detail. Whenever possible *lactic acid milk* should be used as the basis of the feeding. The feeding may be started with fat-free lactic acid milk to which small quantities of carbohydrates have been added. This may be followed by a feeding with equal parts of whole and fat-free lactic acid milk.

In mild cases feedings of 60 mils (2 ounces) eight times daily, and in severe cases ten or twelve feedings of 30 mils (1 ounce), may be given in twenty-four hours during the first two days.

Gradually the fat-free may be replaced by whole lactic acid milk until these infants receive one-fifth or one-sixth of the body weight of this food per day. In very young infants the change to whole lactic acid milk must of necessity be somewhat slower.

The carbohydrates are usually added in a slowly fermentable form, such as the maltose and dextrin compounds, which are usually started by the addition of four grams per kilogram (two grams per pound) and increased until eight grams or more per kilogram of body weight are added. The flour ball or dextrinized barley flour may be used to further supplement the carbohydrates by the addition of an amount equal to one-fourth of that of the sugar added.

Marriott has more recently recommended the use of commercial "glucose," otherwise known as "corn syrup," which is a mixture of dextrin, glucose and maltose, as an available form· of carbohydrate in use in these cases. Of the total carbohydrates present in corn syrup dextrin makes up approximately 55 per cent., maltose 30 per cent., and glucose 15 per cent. He starts the diet by the addition of 3 per cent. to the lactic acid milk and if no diarrhea occurs, he increases it gradually, depending upon the infant's tolerance and the amount of food necessary to cause a gain in weight. In some infants the amount of added sugar may advantageously be as high as 10 per cent.

Infants may be fed on these corn syrup, lactic acid milk mixtures for an indefinite period of time. As a gain in weight occurs the amount of sugar may be advantageously diminished. In infants approximating one-half of their normal weight it may be necessary to feed as much as 160 to 200 calories per kilogram before there is a marked increase in weight. Several weeks of careful feeding may be required before these amounts can be approximated. The whole lactic acid milk and corn syrup mixtures owe one of their chief advantages to the fact that they are a concentrated food and if the infant can take only. a limited number of ounces at a feeding and only a limited number of feedings in a day, the requirements for a high caloric intake can be solved by the administration of such a mixture. A mixture made from whole milk to which the corn syrup is added will have a food value varying from 25 to 30 calories or more per ounce, depending upon the amount of the syrup added.[1]

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[1] Karo—blue label syrup contains from 80 to 85 per cent. of carbohydrate by weight, as its specific gravity is high (approximately 1.40), it contains from 110 to 120 per cent. of carbohydrate by volume. The thick syrup is somewhat difficult to handle and to mix with milk. It is more convenient to prepare a diluted syrup. Mixing 45 volumes of the thick syrup with 55 volumes of water gives a thin syrup containing approximately 50

The individual meals should be increased so as to meet the infant's caloric needs as rapidly as the condition allows.

The need of the athreptic infant for food containing sufficient vitamines can be demonstrated clinically by the rapid improvement noted upon the proper rounding out of the diet. For this reason the infant should not be kept for too long a time on a food deficient in this substance. Cod-liver oil should be added to the diet, beginning in small amounts, as soon as the infant's condition will warrant it. Orange juice should also be started early and increased until one ounce or more is taken daily.

Good results in the treatment of athreptic infants are obtained by feeding them with *albumin milk*.

The advantage of the treatment with albumin milk consists in the fact that it is possible to reach sufficient feeding quantities more rapidly than with most other artificial foods, without the danger of exciting anew the fermentative processes. Thereby the danger of inanition is avoided and reparation is accelerated.

In the *mild* cases of athrepsia we start after an interval of six hours on tea, with administration of 300 mils of albumin milk, with an addition of 3 per cent. of maltose-dextrin preparations (milk-sugar is not advisable, and even the cane-sugar is not so reliable), divided into five or six meals, and with further addition of tea. In the days that follow, without paying any attention to the stools, the quantity of albumin milk is increased, every other day by 100 mils. In the presence of firm stools it is increased even more rapidly, until a daily quantity of 180 to 200 mils per kilogram (3 ounces

per cent. of carbohydrate. One hundred c.c. of this by volume may be considered as containing 50 Gm. of carbohydrate. Such a thin syrup is measured in a graduate and added to the whole lactic acid milk. The mixture should not be agitated sufficiently to separate the fat as butter. The mixture is not further sterilized, but is kept in a refrigerator until used. As such mixtures are very thick, a nipple with a large hole must be used in feeding.

per pound body weight) is reached. A total daily quantity of 1000 mils of albumin milk is rarely to be exceeded. In typical cases dry fat-soap stools appear after one to two days, this is followed by cessation of weight loss, and reparation proceeds undisturbed.

After the quantity of food necessary to sustain the infant is reached, sugar may be gradually increased from 3 to 5 per cent. Dextrinized starches in the form of flour ball (imperial granum), or dextrinized barley flour in quantities of 1 or 2 per cent. of the mixture, can often be added, to advantage, to albumin milk.

In *severer* grade of athrepsia the intestine is allowed to empty itself by a short period of hunger. In spite of the danger of inanition, six, or at most twelve, hours on tea cannot be avoided. This is to be followed by the administration of albumin milk, best by frequent meals (eight to ten), on the first day 200 to 300 mils, and then, as previously advised, rapid increase with gradual diminution of the number of meals and increase in the addition of carbohydrates. If the initial loss in weight does not stop within three to four days, and if the child shows languor and tendency to subnormal temperature, then the addition of carbohydrates must be increased, even in the presence of frequent stools, until the loss stops.

If we proceed in this way, then the number of unsuccessful cases becomes considerably less. Experience has shown that in albumin milk therapy often an error is made which frequently leads to failure by underfeeding. It should be remembered that albumin milk has a caloric value of only about twelve to the ounce, and therefore this feeding must be carefully guarded to avoid: (1) Too slow initial increase, thereby prolonging inanition; (2) omission of carbohydrates or insufficient increase of the same; (3) repeated restriction of the quantity of the food, or withholding carbohydrates when the temperature rises or diarrhea reappears. All these are to be avoided. Only when sudden loss in weight

and violent diarrhea set in, should the total quantity of the food be reduced. After disappearance of these acute symptoms the increase must be made as soon as possible. -

In the beginning of the treatment with albumin milk, exacerbations similar to those that occur on feeding with human milk may occur, and these should not lead to starvation. Later, the gain is rapid, provided that sufficient quantities of carbohydrates have been added.

The duration of feeding with albumin milk is about six to eight weeks for the younger infants, and four to six weeks for the older infants. After this time the conditions change usually to such an extent that ordinary milk mixtures, corresponding to the child's age and weight may well be taken. The change is best made by replacing all the feedings of albumin milk mixtures at one time by a weak boiled milk mixture. This is frequently followed by bad stools for a day or two, which should not lead one to discontinue the new diet. The quantity, however, should not be further increased until they show some improvement.

If a relapse occurs, then it is necessary to return to feeding with albumin milk for some additional time.

One may speak of a complete cure of this nutritional disturbance in an infant only when, after discontinuation of albumin milk and return to the usual milk mixtures, with careful dosage, the development proceeds without any disturbance.

Medicinal Treatment. This is practically limited to stimulation in the presence of collapse and sinking spells, and the favorite stimulant is camphor given intramuscularly in the form of a sterilized camphorated oil (five to ten drops every two to four hours). Aromatic spirits of ammonia and caffeine sodium benzoate are valuable. Epinephrin, in doses of three to five minims of $\frac{1}{1000}$ solution, administered intramuscularly, may be used in an emergency in repeated doses. In the severer types blood transfusion is also indicated,

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After-treatment. In extreme cases intravenous injection of glucose solution may be used to advantage. It increases, at least temporarily, the volume of the blood, and as a result the volume flow through the organs and at the same time furnishes a limited amount of food which may be of considerable value in extreme cases.

Glucose may be administered intravenously in 10 per cent. solution, in amounts not exceeding 20 mils per kilogram of body weight. It is best given in normal saline the total amount not to exceed 100 to 200 mils. ($\frac{1}{50}$ of the body weight). It may be preceded to great advantage by giving 150 to 300 mils of Ringer solution intraperitoneally one hour before. The injection should be given very slowly, and may be repeated two or three times a day.

Artificial heat must be applied in all cases with a decided tendency to low temperatures. This must not be overdone, since the child's temperature can easily be raised above the normal, and act as unfavorably as subnormal temperature.

CHAPTER VI.

ANHYDREMIA (ANHYDREMIC INTOXICATION).

Synonyms: Alimentary intoxication (Finkelstein). Alimentary toxicosis (Czerny). Gastro-enteric intoxication (Holt). Catarrhal enteritis, ileo-colitis, infectious diarrhea, cholera infantum, summer diarrhea.

The term anhydremia (an-privative, hydro-water, aima-blood) means the opposite of hydremia, an abnormal decrease in the relative proportion of the water to that of the salts of the blood. This term, suggested by Marriott,[1] is used to describe a general toxic state precipitated by an insufficient water intake or an excessive loss through diarrhea and vomiting, and by way of the skin and lungs, and resulting in a negative water balance and a diminished blood volume. It is characterized by a symptom-complex in which diarrhea and irritability of the central nervous system are the most characteristic signs. The collapse and nervous symptoms may outweigh the intestinal symptoms. Less frequently cases may be seen unassociated with diarrhea and which are due to insufficient fluid intake.

The graver the preceding nutritional disturbances, that is, the closer the infant approaches the stage of athrepsia, the more readily does the stage of anhydremic intoxication develop.

Etiology. The symptom complex has as its basis a desiccation of the blood and tissues, the result of an increased excretion of water over the intake. The end

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Note[1]: In the elaboration of Chapter VI free use has been made of the original articles of W. McKim Marriott. See Pathogenesis of Certain Nutritional Disorders, Proceedings of American Pediatric Society, xxxi, 1919; Some Phases of Pathology of Nutrition in Infancy, Am. Tour. Dis. of Children, xx; 461, Dec. 1920; Severe Diarrhea in Infancy, M. Clinics, N. Amer. iv, 717; November, 1920.

results of this abnormal metabolism is a characteristic group of symptoms which are in large part directly attributable to the concentration of the blood and its effect upon the function of the other organs.

The group of symptoms are suggestive of an acute poison which may result in death and in the majority of instances leave no demonstrable pathological findings. In the absence of anatomical changes we are forced to conclude that some profound alteration in the chemical or physiological processes in the body must have occurred. Czerny, who gave this clinical picture considerable thought, believes that it was the result of the absorption of toxic substances of metabolic or bacterial origin. Finkelstein believed that it was primarily due to food poisoning and that infection and constitutional abnormalities acted as contributory factors to the lowering of the infant's tolerance for food, thereby impairing his ability to utilize food without the production of harmful substances. They base their conclusions upon the fact that the clinical picture is aggravated when a relative excess of food is given to this class of infants. Marriott, in his analysis of this group of cases, recognizes the bad effect of food when given in excess of the infant's tolerance but explains it upon a different basis. He emphasizes the well known fact that infants suffering from diarrhea have a tendency to become worse when food is given, especially an excess of carbohydrate or fat. The result of this is an increase in the degree of anhydremia. If food is given to an infant who is anhydremic, such as a case of athrepsia, even though diarrhea is not present, it is likely to result in diarrhea. It is therefore evident that any of the etiological factors which may cause or predispose to diarrhea may be the forerunners of the stage of anhydremia, due largely to the loss of fluids through the bowels and to a lesser degree through vomiting. The secondary picture of acidosis may be superimposed upon that of anhydremia

with clinical evidence of an intoxication. The acidosis in these cases is due to a deficient renal function secondary to the impaired circulation through the kidneys and results in an accumulation of acid phosphates in the blood.[1] A second factor may be active in the development of the acidosis, in that there is a tendency toward accumulation of lactic acid in the tissues from partial cell asphyxia which is also dependent upon the diminished blood flow secondary to a diminished blood volume. Uremia may be superimposed in this stage. We believe that we are justified in assuming with Marriott that the absorption of unsplit sugars into the blood through injury of the intestinal mucosa and the products of fat metabolism, as suggested by Finkelstein, are not the underlying etiological factors. However, sugar may increase the symptoms of intoxication through increasing the diarrhea and water loss. Fats and salts may have a similar action. Sugar further furnishes a good culture medium for the growth of abnormal bacteria and for the normal intestinal bacterial flora which under abnormal conditions may inhabit the upper intestinal tract. The abnormal flora may enter from above through infected food or ascending from below to higher levels, they may invade the upper area in the small intestines in abnormal numbers, due to diminished resistance of the individual. Again, in disturbed metabolism an excess of sugar may reach the lower bowel and stimulate abnormal bacterial activity. It is also possible that the protein split product (amino-acids) may give rise to abnormal amounts of histamine and allied substances which are very irritating to the intestinal mucosa and thereby cause or aggravate the diarrhea. It is therefore evident that all factors which cause nutritional disturbances can be active factors in the causation of a state of anhydremia with its

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1 Marriott, W. McKim and Howland, J.: The influence of acid phosphate on the elimination of ammonia in the urine, Arch. of Int. Med., xxii, 477, 1918.

secondary picture of intoxication. Although frequently the underlying diarrhea is due to a primary food disturbance the diarrhea is more commonly seen following a food disturbance secondary to some other factor.   -

Anhydremia also frequently occurs in the absence of diarrhea when the amount and character of the food is not such as to cause a gastro-intestinal disturbance and as a secondary complication in infections. It is especially common in premature infants who are not given sufficient inert fluids between feedings, in young infants nursing dry breasts and in the presence of anorexia in the course of acute illnesses. It is a common condition among infants suffering from intra-cranial lesions, such as intra-cranial hemorrhages, and idiots. Also in the course of acute infections, such as pneumonia, otitis media and pyelitis, more especially during the acute febrile stages associated with delirium. In all of the foregoing, vomiting may be an active factor.

Desiccation of the body may therefore occur in a great variety of conditions and the symptoms are essentially the same, no matter what the original cause, and while diarrhea is by far the most frequent etiological factor, it need not necessarily be present. In the light of our present knowledge the terms "anhydremia" and "anhydremic intoxication," as suggested by Marriott, are accurately descriptive, the former of the underlying factor and the latter when the toxic stage has been reached, irrespective of the original cause.

Symptoms. *Fever.* A rise in temperature is one of the first symptoms. It may be slight, or it may go up to 104° or even 106° F. The height of the temperature is not always a direct indication of the severity; in fact, the several types associated with athrepsia may have a low temperature. Prompt withdrawal of the food in cases unaccompanied by infection is usually as quickly followed by a lower temperature when at the same time there is sufficient water administration and retention.

However, if the offending food is continued, we soon have other symptoms suddenly and to an alarming degree. The weight of evidence seems to strongly support the view that the action of the administration of hypertonic solutions of sugars or salts is to remove water from the body with resulting further dehydration. The assumption of Finkelstein, that the continuing of the diet resulted in the production of pyrogenic metabolites, is not tenable. Fever as the result of dehydration has been repeatedly observed, such desiccation resulting in interference with water evaporation through a decreased circulation and secondarily by interference with excretion. The second factor of infection leading to diarrhea and vomiting, and dehydration of the body is an added causative factor in the explanation of the fever.

Rapid *loss in weight,* even one to two pounds in a few days. This is mainly due to loss of salts and water.

Vomiting is frequent and may contain blood if long continued.

The stools are liquid, usually numerous, and contain mucus, and occasionally blood. In the severest cases—cholera infantum—the stools assume a rice-water appearance, move almost continuously, and are often associated with tenesmus, and not infrequently prolapse of the rectum. Exceptionally, an obstipation is seen in place of the diarrhea, and when this is associated with vomiting and abdominal distention, one cannot help but think of intestinal obstruction.

The *general appearance* of the patient changes. The skin is gray in hue, and becomes wrinkled; the eyes are sunken, with distant stare, and the nose assumes a pinched appearance. The skin hangs in loose folds; it is dry and has lost its elasticity so that it may be picked up into ridges which remain an appreciable interval before flattening out. The lips are dry, parched and often of a peculiar cherry red color. The mouth is held partly open, the tongue is dry.

*Nervous symptoms and psychic disturbances are usu-
ally pronounced,* and often lead to a confusion with
meningitis. The infant is restless; the sensorium is dis-
turbed, with an occasional cry as if in pain. Before
these more severe symptoms develop, the child appears
apathetic, drowsy, and dopy. The face assumes a fixed
expression, and there is a tendency on the part of the in-
fant to lie constantly in one position, and when the child
moves its extremities it does so slowly, as if too tired or
weak to change its position. The arms are not infre-
quently flexed in an attitude resembling that of a prize
fighter. If the condition increases in severity, stupor and
coma, associated with twitchings, convulsions, strabismus,
and other meningeal symptoms, ensue.

The respiratory manifestations may vary from a slight
increase in number and depth to a marked dyspnea. The
respirations are often of the "air hunger" type, such as
is observed in diabetic or uremic coma (deep, rapid and
without pause). The breathing becomes both costal and
abdominal, the whole thorax rises with each inspiration
and accessory muscles are brought into play. This is
due to acidosis, which is, however, not the result of an
overproduction of acetone bodies, but, as demonstrated
by Howland and Marriott,[1] it is caused by a failure of
the kidneys to excrete acid phosphate and in part to acids
produced in the tissues as a result of diminished oxida-
tion due to the deficient blood circulation.

The urine is small in amount, even to anuria. It may
contain albumen and not infrequently casts. The kidneys
become functionally inactive, although there are usually
no demonstrable pathological changes. This alteration
in the functional capacity of the kidney results in the
accumulation in the blood of products ordinarily elimi-
nated by the urine. Uremic symptoms are not infre-
quent as a result. Occasionally glycosuria is present.

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[1] Howland and Marriott: Am. Jour. Dis. of Children; 11,
309, March, 1916.

Schloss[1] found the sugar in the urine to be either glucose alone or glucose in combination with galactose or lactose. A certain amount of lactose can pass through the intestinal mucosæ and it may well be the case that when fairly strong solutions of lactose are introduced into the intestinal tract of these infants that some would be absorbed unchanged. As lactose is not altered outside of the intestine, it would be excreted quantitatively in the urine. It has been shown by Araki[2] that asphyxial conditions occurring as the result of vasoconstriction, hemorrhage, or a diminution of the oxygen carrying capacity of the blood, lead to glycosuria. This is generally supposed to be the result of increased glycogenolysis, dependent upon acid production in the tissues. In the case of these infants the glycosuria may be readily explained on the same basis.[3] Schloss has demonstrated an excess of urea and total non-protein nitrogen in the blood, reduced phenolsulphonephthalein excretion in the urine and an abnormally high Ambard co-efficient as evidence of impairment of renal functions. He also found that the extent of renal function involvement was proportionate to the degree of desiccation of the blood.

The *Heart:* Action is weak and the *pulse* is small, often rapid and irregular.

The *Blood* presents changes which are characteristic and constant, varying only in degree. The changes are dependent upon the severity of the case. In the severer cases the blood is obtained with difficulty due to the fact that it is thick and does not flow easily, and when centrifuged separates a relatively small amount of serum. These findings are due to the fact that it is concentrated by water loss. The specific gravity is increased, as is

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[1] Schloss, O.: Am. Jour. Dis. of Children, xv, 165, 1918.

[2] Araki: Ztschr. Physiol. Chem., Series of articles in 15, 16, 17, 19.

[3] Marriott: Am. Jour. Dis. of Children, xx, 461, 1920.

also the index of refraction. There is a negative nitrogen and salt balance. The viscosity, the electrical conductivity and the osmotic pressure are all increased. The state of uremia, so frequently seen in the fatal cases, may be a direct result of the increased colloidal osmotic pressure of the blood over the arterial pressure in the renal glomeruli, which results in decreased secretion of urine by the kidneys. Diminished blood volume results in a greatly diminished volume flow of the blood. Therefore, less blood flows through a given portion of the body in a unit of time. This leads to an accumulation of acid products of metabolism in the tissues and a decreased alkali reserve of the blood, that is to say, acidosis. The blood flow in the arms of normal infants, measured by the Stewart method; by Marriott, ranged from 15 to 22 mils per 100 Gm. per minute. In some infants suffering from anhydremia he found it as low as 2 or 3 mils per minute. The blood pressure is usually maintained through the high blood viscosity. A comparison of the red blood cell counts, made on venous and capillary bloods, shows a marked concentration of corpuscles in the capillaries. This stagnation of red blood cells in the capillaries, as the result of arteriolar constriction, may explain the peculiar gray pallor of the skin, which tends to disappear with the establishment of a normal circulation. The leucocytosis, which is almost invariably present, may in part, at least in the uncomplicated cases, be due to a damming back of the leucocytes in the capillary blood. In those complicated by infection a higher count is often seen, ranging from 15 to 35.

Sclerema is present in the severer types—a very bad sign—due to a coagulation of tissue fluids of an unknown nature.

Enlargement of the liver accompanies the severe types.

Pathogenesis. As has been stated, a great deal of thought has been given to the clinical picture which has

been described, both in American and European clinics. There has been a considerable difference of opinion as to the underlying factors influencing the train of symptoms. In the well-developed case a large number of symptoms are regularly and coincidentally present and it has been the object of the various clinicians to find an adequate explanation for all of the symptoms. The clinical picture in those infants who have lapsed into a toxic condition following severe diarrhea may be explained on the basis of the *water and salt losses from the body*. In all probability there is also a deep-seated change in the water binding functions as a result of the loss of salts and the breaking down of glycogen necessary to meet the metabolic functions during the period of under-feeding. Unfortunately, this increased water and alkali loss by the intestines is not balanced by lessened kidney secretion until the renal function itself is impaired through circulatory disturbances. There is also an increased water loss through the lungs and temporarily at least in the presence of fever, through the skin. Once anhydremia has occurred "a vicious circle" is established which still further results in lowering the functional capacity of the gastro-intestinal tract. Even in the absence of diarrhea there is an interference with the functional capacity of the intestines, with a resulting increased decomposition of food, the products of which may cause further injury to the intestine itself. Following this there may be an absorption of bacterial poisons from the intestines which may result in a toxic catastrophe. When diarrhea is present the administration of food has a tendency first to irritate the intestine, more especially when hypertonic sugar solutions are administered; salts, fats and proteins may have a similar action. The absorption of the soluble metabolites, by their accumulation in the blood, increases its osmotic pressure and thereby decreases the available water reserve. Marriott believes that these facts are an adequate explana-

tion of Finkelstein's observation, that the giving of food increased the symptoms of his infants suffering from alimentary intoxication. It is quite likely that interference with the function of the liver becomes an important factor and the absorption of its by-products may aggravate the fever and diarrhea.

The secondary picture of *acidosis* may be superimposed upon that of anhydremia with clinical evidence of an intoxication. The acidosis in these cases is due to a deficient renal function secondary to the impaired circulation through the kidneys and results in an accumulation of acid phosphates in the blood. A second factor may be active in the development of the acidosis, in that there is a tendency toward accumulation of lactic acid in the tissues from partial cell asphyxia which is also dependent upon the diminished blood flow secondary to a diminished blood volume. Uremia may be superimposed in this stage. The state of acidosis is responsible for the "air hunger" type of respiration.

Marriott and Perkins made a series of investigations to ascertain the presence of a negative water balance in two groups of cases, the first unassociated with diarrhea and the second with diarrheal disturbances, to prove that the symptoms in both groups were associated with similar blood findings. As a criterion of the degree of desiccation of the body fluids they determined the index of refraction of the blood serum. This determination is easily made and requires only one or two drops of serum. It has the advantage that it may be repeated at frequent intervals. The refractive index varies with the concentration of solids in solution. As the protein has much greater effect on the index than the other solid constituents of the serum, it is possible to determine quite accurately the protein content of the serum by means of the refractometer. The protein content of the serum of normal infants during the first six months of life averages about 6 per cent.—toward the end of the first

year it is often as high as 7 per cent. [1] [2] Marriott and Perkins[3] observed an increase in the protein content of the serum of as great as 50 per cent. above the normal for the age. They were able to demonstrate a state of anhydremia in conditions unrelated to diarrhea, such as pneumonia, otitis, pyelitis and non-infectious conditions in which food is refused, as well as in diarrheal disturbances.

Pathology. In the small intestine there is usually no marked change. Hyperemia of the mucous membrane and enlarged follicles, especially Peyer's patches, are usually present. The liver and kidneys show a hyperemia, cloudy swelling, and fatty degeneration (probably causing hepatic and renal insufficiency). Other tissue changes which may be present are not specific but such as might be expected from similar noxæ acting at any time.

Diagnosis. The diagnosis is based on the above symptoms, and improvement on withdrawal of food, in the presence of a sufficient water administration by mouth, subcutaneously, intravenously or intraperitoneally. The most characteristic and striking symptoms are those of the nervous system resulting in stupor, pauseless respirations, and a toxic appearance. These are usually associated with diarrhea, vomiting and a rapid loss in weight. The history of preceding nutritional disturbances and infections are of great importance in diagnosis.

Prognosis. The prognosis in anhydremia depends upon the underlying causes, the degree of desiccation and the previous nutritional condition of the infant. When anhydremia is due simply to insufficient fluid intake it usually clears promptly after a sufficient amount of water has been taken by mouth. Anhydremia resultant from diarrhea or the combination of vomiting

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[1] Reiss: Jahrb. f. Kinderh., lxx, 311, 1909. Ergeb. d. Inn. Med. u. Kinderh., x, 531, 1913.

[2] Salge: Ztschr. f. Kinderh., 1911, pp. 126, 317.

[3] Personal communication.

and diarrhea is more serious than that due to other causes. The prognosis is especially bad in the case of infants already athreptic. The presence of acidosis renders the prognosis exceedingly grave, not so much on account of the acidosis itself, for that can be cured, but because the presence of acidosis indicates that extreme desiccation has occurred, with profound disturbance of the metabolism.

As infections, both parenteral and enteral, are commonly the underlying causative factor, the further dangers from this source must not be under-estimated and further treatment must be directed to their relief. The prognosis is also directly dependent upon the ability of the infant to retain a sustaining diet after the subsidence of the acute toxic symptoms. The younger the infant, the more difficult will be the problem unless breast milk be available.

Treatment. In the presence of vomiting and diarrhea, more especially when they are complicated by an inability to retain sufficient inert fluids, every effort should be made early in their treatment to prevent the development of anhydremia. Vomiting and diarrhea are to be treated as previously recommended. (Page 230).

All food should be withdrawn for from six to twelve hours and in some cases as long as twenty-four hours.

Sufficient water must be administered to overcome the loss from all causes. The average infant, in the presence of vomiting, diarrhea and fever, will require a minimum of one-fifth of its body weight in water in twenty-four hours. In order to estimate the amount administered, a careful record must be kept of the amounts given and retained.

As much as possible should be given *by mouth* in small feedings at frequent intervals. Water given immediately after vomiting is frequently retained.

A single gastric lavage is indicated if food has been given shortly before or in the presence of repeated vomiting.

In severe types *subcutaneous* injection of saline solutions, two or more times daily, are indicated, 100 to 300 mils can be injected each time. Ringer's solution may be used to advantage for this purpose.

	Gm. or Mil.
NaCl	7.5
KCl	0.1
CaCl	0.2
Water	1000.0

The water used in making this solution should be redistilled shortly before using.

The same solution may be administered *intravenously* in young infants through the longitudinal sinus and in older infants into the anterior jugular or median basilic vein. It should, however, be remembered that there is danger of collapse from acute cardiac dilatation when excessive amounts are introduced rapidly into the circulatory system. On the average an amount not to exceed $\frac{1}{50}$ of the body weight should be injected at one time. The gravity method should be used. The danger of administering fluids by way of the longitudinal sinus must be appreciated.

Probably the most efficient means of introducing water into infants of this type is by way of the *peritoneal cavity*. This method of administration was first used extensively in this country by Howland,[1] after seeing its successful application by Garrod, in London.

Large amounts of fluid may be given this way and be rapidly and completely absorbed. Ringer's solution is the best for this purpose, and should be freshly sterilized before using. The injection is easily given and causes very little pain or discomfort. The needle used for the

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[1] Blackfan, K. D. and Marcy, K. F.: Am. Jour. Dis. of Children, vol. xv, 19, 1918.

injection should not be very sharp. A suitable size is 18 gauge, which is about the size commonly used for serum injections. Strict aseptic technique is absolutely essential. There is but little danger of puncturing the intestine, if the method described is carefully followed. This route of fluid administration should not be undertaken unless all details can be fulfilled.

The technique is as follows: If abdominal distention is present, this must first be relieved. The patient lies in the recumbent posture, with the movements of arms and legs restricted by a restraining jacket. The most favorable site for the introduction of the needle is through the linea alba, just below the umbilicus: The skin is prepared after the usual surgical method, with iodin and alcohol, and the area may be rendered anesthetic with ethyl chlorid. The solution is introduced by gravity from an infusion bottle, only slightly elevated. It should be introduced at about 100° F. When the patient is restrained in the proper position and the site for the injection prepared, the skin and subcutaneous tissues are picked up between the thumb and the index finger, and the needle, pointing upward, is inserted at an oblique angle. After it has penetrated the peritoneum the fluid is allowed to flow into the peritoneal cavity.

The injection of the fluid is continued until the abdomen becomes slightly distended. It should not be introduced too rapidly or in too large quantities, to avoid the possibility of embarrassing the respiratory and circulatory systems. Two hundred to 500 mils can be introduced in from fifteen to twenty minutes. After the fluid has been introduced, the needle is withdrawn and the puncture wound covered with a sterile dressing. The injection may be repeated after six to twelve hours, if necessary. With repeated injections, even with an isotonic solution, there develops an edema of the peritoneum and frequently some exudate. This disappears without untoward effect. However, with the use of hypertonic

solutions more serious injury of the peritoneum may result. We have therefore discontinued the use of glucose and bicarbonate of soda in the solution.

If the infant presents evidence of *acidosis,* dextrose should be added to the saline solutions and administered intravenously. Six Gm. (90 gr.) of dextrose may be added to 120 mils (4 ounces) of saline solution and repeated in four to six hours, if indicated. Pure dextrose is essential. Only rarely is there an indication for the administration of alkali. It should be remembered that intravenous administration of larger amounts of sodium bicarbonate may result in collapse. (See Acidosis.)

Saline per rectum is best administered by the drop method unless the infants are too restless. Thirty drops per minute for four hours is 450 mils. One-half strength of Ringer's solution may be used. Sodium bicarbonate, 5.0 Gm. (75 grs.) may be added to every 500 mils of the solution (1 per cent.).

It is necessary that sufficient administration of water be continued until the causative factor of the anhydremia is no longer operative. The initial loss in these infants may reach amounts approximating 1000 to 2000 grams.

The treatment must also be directed to the overcoming of any infectious processes which may be present. It is to be rememberd that the severe forms encountered are often seen in infants who have suffered from repeated attacks of nutritional disturbances, and that in the presence of athrepsia the convalescence must necessarily be slow. All laxatives are to be avoided in the presence of marked diarrhea, as the bowels empty themselves and any further purgation increases the loss of salts and water and the tendency to the development of an acidosis.

Opium is indicated when the stools are frequent, large and watery, and remain uncontrolled by other methods. Paregoric in suitable doses per mouth, or the tincture per rectum, may be used with care.

Analeptics. Give a mustard bath in case of collapse. Reddening of the skin is a good sign.

Antipyretics. Use tepid packs, and leave the infant undressed. Ice-cap to head is useful, but should not be applied directly to the head, because of the thinness of the skull in young infants.

Stimulants. In collapse, warm packs or baths are indicated. Caffein sodium benzoate, 0.006 Gm. to 0.030 Gm. (0.1 to 0.5 gr.), four or five times daily; camphorated oil in 1-mil doses every two hours hypodermically if indicated; epinephrin solution, 0.2 to 0.3 mil (1 to 1000), subcutaneously or intravenously.

Sedatives for Convulsions. Sodium bromide, 0.2 Gm. to 0.3 Gm. (3 to 5 gr.), repeated in three to four hours; veronal, 0.05 Gm. (1 gr.). Chloral hydrate is best avoided.

An electric fan is a most valuable addition to our therapeutic measures in summer.

Lumbar puncture may be indicated in the presence of increased intracranial pressure, and for diagnostic purposes.

Diet. Hunger diet should be employed rarely longer than twenty-four hours. When infant is stuporous, water should be administered by gavage at regular intervals of about three to four hours.

In cases of food intoxication, twenty-four hours on a hunger diet, with sufficient water, causes striking changes. The child looks bright, smiles, and to all appearances looks convalescing, notwithstanding a usual loss of weight. The stools also become less frequent, and although small and containing mucus (hunger stools), they cause less irritation of the buttocks and little loss of water. The improvement is no less striking than that seen in the crisis of pneumonia.

Human Milk. Human milk is, by all means, the best food. Feed often, and in small amounts, ten times daily, five mils from bottle or spoon. The infant may also be

placed directly at breast for one- or two-minute periods, in less severe cases. Increase when the temperature, etc., does not react to food, and then not more than 50 to 100 mils daily increase at first. After several days, if the infant shows no evidence of relapse, it is again placed unrestrictedly on the breast. If this is done too soon, relapses occur. A too prolonged starvation adds the danger of inanition.

A sustaining diet should be reached in eight to ten days (70 calories per kilo), after which the child can be put on the breast five times daily. Weigh infant before and after feeding, if placed at breast. The gain in weight is often slow in the stage of repair on human milk, due to the low protein and salt content.

Cow's Milk. The feeding of these infants, for whose use breast milk is not available, should follow the same principles as outlined for the treatment of diarrhea. (See page 231.) It must, however, be remembered that even greater care is needed because of a tendency to recurrence of acute diarrheal attacks. In every case where there is a second recurrence of toxic symptoms, breast milk is absolutely indicated. These infants do best when they are fed with small quantities—10 or 15 mils—repeated at short intervals, with the administration of eight or ten feedings daily. This holds true with the feedings of albumin milk, as well as with the fat-free lactic acid and skim milk plus gruel mixtures. At all times the water administration should be held at the maximum. For the first few days after the hunger day, a food low in fat and sugar should be fed. Even on this low diet weight loss usually stops but this should not lead to an underestimation of the great danger from a too prolonged under-feeding. These infants offer every indication for the use of our best judgment.

CHAPTER VII.

INFECTION AND NUTRITION.

THE intimate relation between infection and nutrition may be made clear by considering the subject under three headings:

1. The susceptibility to infections as influenced by previous diet and the state of nutrition.

2. The course of infections as affected by diet and the state of nutrition.

3. The influence of infection upon nutritional processes.

      (*a*) Parenteral infections.

      (*b*) Enteral infections.

1. Susceptibility Influenced by Nutrition.

The previous diet and the state of nutrition being the same, there are marked individual differences in the susceptibility to infection. Among the breast-fed infants there are on one hand infants who remain free from any infection, even under very unfavorable external conditions, while on the other hand there are breast-fed infants who under favorable conditions often contract an infection. This points to congenital differences based on the difference in the *constitution* of the individual. As a rule, the lowering of immunity is not the only sign of inferior constitution in these infants, but they show a number of other symptoms of a constitutional anomaly, such as exudative and neuropathic diathesis. In this group of infants the susceptibility to infection becomes even more striking when they are put on artificial feeding, and especially when the diet is improper. In infants with constitutional anomalies one is justified in thinking of an abnormal composition of the tissues and of the

(*276*)

body fluids, both the latter factors in themselves leading to a lowering of immunity.

The natural immunity of the healthy breast-fed infant affords the best example of the importance of the *diet* in the establishment of resistance to infection.

In the artificially fed infants the increased susceptibility to infection is usually based on nutritional disturbances, which, however, may be so slight as to escape recognition. However, when a careful study is made of the feeding history the cause can usually be demonstrated in a poorly balanced diet, more commonly one .excessive in carbohydrates and fats, which result in an abnormal composition of the tissues (see Nutritional Disturbances). Those modes of feeding which cause normal tissue chemistry diminish susceptibility, while every form of feeding which unfavorably influences metabolism increases susceptibility to infection.

In artificially fed infants these facts offer valuable therapeutic suggestions, and should lead one to avoid overfeeding as a whole as well as of the individual constituents of the diet, and the early administration of the mixed diet.

The susceptibility to infection is increased by every nutritional disturbance. This applies to the simple and seemingly harmless digestive disturbances, as well as to the more severe forms. (Athrepsia, anhydremia.)

2. Course of Infections Influenced by Nutrition.

The course of the infection is essentially influenced by constitution, age, hygienic conditions, mode of feeding, and the state of nutrition. The premature and the very young react poorly to infections. Gastro-intestinal, pulmonary and septic infections of the newborn have usually an unfavorable course, especially in the artificially fed infants. Infants suffering from constitutional anomalies are less likely to react favorably than normal, healthy infants. In all infants suffering from exudative

or neuropathic diathesis even slight infections should be given serious consideration.

Nutritional disturbances have a direct influence on the prognosis of all forms of infections. This is more especially true of the infections of the respiratory passages, in which a simple rhinitis or pharyngitis may readily be complicated by pneumonia and severe gastro-intestinal complications, but also true of the simple skin infections, which may rapidly take a serious course resulting in sepsis.

The institution of a proper diet is of primary importance in all cases of infections.

Feeding with human milk is the treatment of choice. If this is not obtainable, and it is necessary to feed artificial food mixtures, they must of necessity be well balanced, and one-sided carbohydrate diets are to be avoided. Whenever possible, a mixed diet should be instituted.

3. Infection Influencing Nutrition.

Infection may produce any form of nutritional disturbance, from the slightest forms to the most severe forms of athrepsia and anhydremia. For the production of nutritional disturbances. infections are to be ranked as of equal importance with dietetic errors.

Although the course of alimentary nutritional disturbances may be similar to that of nutritional disturbances due to infection, still there are important differences that must be kept constantly in mind in order to avoid failures in the treatment. The following table briefly summarizes the most important differences between the two forms of nutritional disturbances:

Nutritional Disturbances due to Alimentation.	Nutritional Disturbances due to Infection.
History of dietetic errors, especially high sugar and fat feedings or underfeeding.	Acute disturbances not so much dependent on the nature of the diet.

Nutritional Disturbances due to Alimentation.	Nutritional Disturbances due to Infection.
Disintoxication of toxic states (fever, nervous symptoms, etc.) by withdrawal of food, and administration of sufficient water.	Toxic states continue or even become worse in spite of withdrawal of food.
Improvement in general condition, and especially of diarrhea, on correction of the diet, especially by reduction of fat, whey and sugar component part.	Persistence of diarrhea after similar change of diet, at least in a number of cases.
Progressive narrowing of food tolerance in untreated cases.	Spontaneous increase of tolerance without special dietetic treatment after the infection passes over (in majority of cases, not always).

(A) Parenteral Infections.

Etiology. It has already been pointed out with what great frequency infants and children suffering from nutritional disturbances are subject to secondary infection. The most frequent of these are those of the skin, respiratory, gastro-intestinal, and genito-urinary tracts, ears and general septic infections.

In contradistinction to this, infections, such as "colds," tonsillitis, pneumonia, otitis, cystitis, pyelitis, which are accompanied by lowered food tolerance, are accompanied by diarrheas. They are likely to run a more severe course than the primary nutritional disturbances.

Undoubtedly many cases of diarrheal disturbances in the course of acute infections are due to an infection of the gastro-intestinal tract following the swallowing of organisms from the upper respiratory tract, thereby developing a secondary enteral infection.

The common occurrence of the *"summer diarrheas"* leads us to search for a relationship between heat and the nutritional disturbances as seen in summer. This rela-

tionship has already been discussed under the chapter on Anhydremia Intoxication. However, it may be well to briefly enumerate the factors which are important in the causation of these nutritional disturbances. High temperatures cause systemic depression, and directly influence all of the body functions. Less food is required in hot weather, and therefore the previous diet may be considered excessive in many instances. Bacterial action on the milk, and the subsequent production of toxic bodies, is a factor of primary importance. An excessive retention of heat by overdressing during the summer months has been proven to be a contributing factor by McClure and Sauer.[1]

A study of the cases of diarrheas in the wards of Sarah Morris Hospital by Gerstley and Day during the course of two summers showed that most of our intestinal cases were secondary to parenteral infections, and not primary intestinal infections, as described by Kendall and Day in their investigations of the Boston epidemics. This could in greater part at least be accounted for by the fact that all of the milk fed to our infants was either pasteurized or boiled, while in the eastern cities considerable raw milk was fed.

Symptoms. By careful clinical observation and experimental investigation we know that infection may produce the following changes:

1. Lessening the gain in weight without any acute symptoms manifesting in the gastro-intestinal canal during or after the infection.

2. Loss of weight and changes in the stools corresponding to the acute nutritional disturbances.

   (a) Acute disturbances of the nature of diarrhea beginning with the infection and disappearing after the infection has been overcome.

   (b) Acute disturbances which begin with the infection, but remain even after the infection is

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1 Sauer: Am. Jour. Dis. of Child., 1915, ix, 490.

overcome, under certain conditions for weeks (chronic intestinal indigestion).

   (c) Grave nutritional disturbances beginning with the infection, but soon becoming the most prominent factor in the clinical picture, with or without toxic symptoms.

Diagnosis. Anhydremic intoxication is usually easily recognized by the nervous symptoms, toxic expression, pauseless respiration, and marked drops in the weight curve. In intoxication, temporary complete withdrawal of food with sufficient water administration in the absence of severe infection results in disintoxication. This is known as *therapeutic dietetic test.* In parenteral infections this is not the case, and starvation only leads to further reduction of resisting power, and therefore should not be long continued.

It is necessary to avoid the mistake of overestimating the intestinal condition for which in many cases the physician is called, and thereby failure to recognize the underlying infection, such as "cold," bronchitis, pneumonia, pyelitis, etc., as a fundamental factor.

Treatment. For treatment practically the most important characteristic of nutritional disturbances due to infection is the spontaneous rise of food tolerance after the cure of the infection.

The primary infection calls for foremost consideration, and its treatment must necessarily vary according to its nature. The intestinal condition, on the other hand, if mild in nature, frequently calls for little treatment in these infants, more especially because in the presence of fever there is a tendency to reduce the intake of food, which in itself is sufficient to correct the intestinal disturbance. Further, with the improvement of the infection the appetite returns, and the infant will demand increased food.

Where it is possible to keep up the baby's nutrition by the proper administration of food during the course of an

infection, such children may be subject to little or no weight loss. In more serious cases the food must be reduced both qualitatively and quantitatively, more especially the sugars and the fats. However, in order to avoid catastrophes, long-continued underfeeding or starvation must of necessity be avoided, since this treatment, causing insufficient nutrition of the body-cells, would decrease the resistance of the infant. Albumin milk, and skim and buttermilk mixtures, with small amount of sugar are best used. Carbohydrates should be added as rapidly as the infant's condition will permit. Prolonged carbohydrate starvation is to be avoided, more especially in the presence of high temperatures and acidosis. They should be increased gradually. In grave nutritional disturbances, with sudden losses of weight and toxic symtoms, complete withdrawal of food cannot be avoided.

In young and weak infants, as previously stated, breast milk may be imperative. In older infants, and those less severely infected, albumin milk, with 2 or 3 per cent. of sugar addition, or buttermilk and skim milk mixtures are frequently well taken. In all cases inanition must be avoided by keeping the child on a sustaining diet of 70 calories per kilogram, or an amount above this.

The type of infants who have been improperly fed, more especially those who have been raised on condensed milk or other foods containing a minimum of fat and protein, but an excess of carbohydrates, offer greater difficulties, because they possess a limited immunity to all forms of infection, beside reacting poorly to changes in their diet during illness. They also react very poorly to starvation, rapidly passing into a state of decomposition. The treatment in these cases should follow that outlined for milder forms of athrepsia.

To repeat, under all circumstances food should be restricted as little as possible.
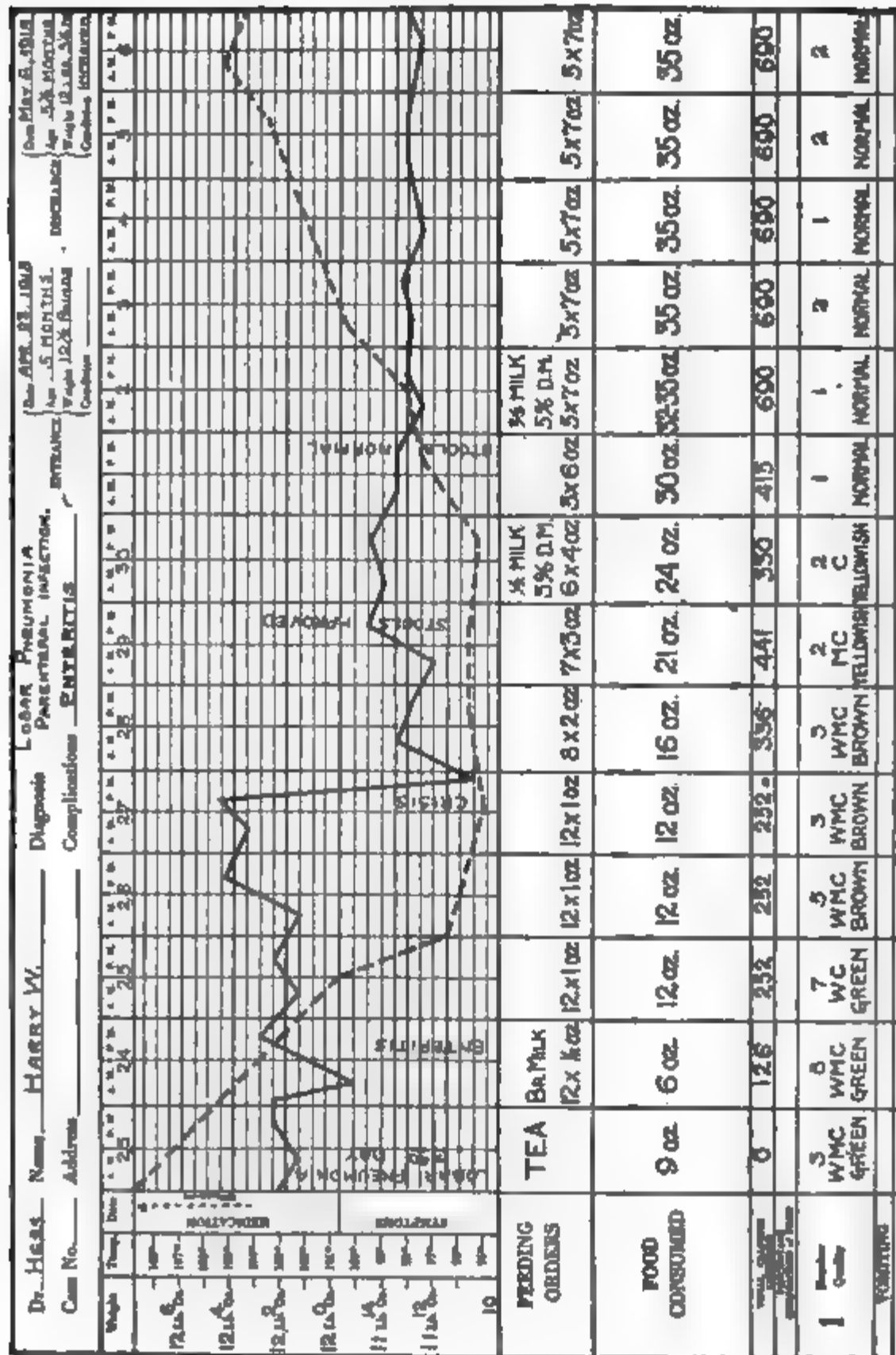
Fig. 11.—Chart showing hospital record of an infant with parenteral infection complicated by enteritis.

The two most important symptoms calling for treatment in the course of parenteral infections are (1) vomiting and (2) refusal of food.

If temporary reduction in food does not relieve vomiting, it may be necessary to resort to gastric lavage which is best performed with 1 per cent. sodium bicarbonate solution, allowing 2 or 3 ounces of the solution to remain in the stomach, with the administration of slightly sweetened tea or cereal waters *ad libitum*, as retained. Prolonged starvation must be avoided.

We have found chymogen milk fed in small quantities at two- to three- hour intervals especially suitable for these cases. This is probably due to the fact that the casein is precipitated in a flocculent form.

Refusal of food which is commonly experienced in these infants calls for catheter feeding (see chapter on Methods of Feeding Premature Infants).

(B) Enteral Infections.

Etiology. Besides the alimentary nutritional disturbances proper, there are in childhood, and especially in infancy, numerous diseases that have to be regarded as true infections of the digestive canal, due to invasion of pathogenic bacteria, or increased and changed activity of the bacteria normally present. In many cases infective material is introduced by food, and especially by the milk, in which the micro-organisms are present, being derived from the diseased cattle that furnish the milk (Streptococcus from inflamed udders, Bacillus coli from feces) or bacteria pathogenic for the human may find their way into the milk in transportation from the place of production to the place of consumption. Besides this, water or contaminated foods other than milk may be the medium through which infection takes place.

There are numerous cases of transmission by contact. These are most commonly seen in the epidemic appear-

ance of gastro-enteritis in institutions for small children and infants. A small, but typical epidemic is reported by Smillie,[1] who had observed it during his study of epidemiology of bacillary dysentery. Four babies developed bacillary dysentery in the wards of the hospital, each of them having been admitted with quite a different diagnosis, and their stools having been negative on admission. Each developed the disease seven to ten days after admission, and in no instance did the infant come from an infected home or neighborhood. Similar epidemics are experienced in all institutions caring for infants, in which aseptic nursing is not practiced.

The environment of the infant, and especially lack of proper cleanliness generally, and in preparation of food especially, are very important factors, which make the enteral infection possible.

Parenteral infections are often followed by enteral infections, and this is especially true of infections of the respiratory tract which often lead to what has been called "bronchoenterocatarrh."

The most important clinical condition among the enteral infections is inflammation of the intestinal mucosa (enteritis), brought about by a variety of bacteria, and accompanied by slimy, purulent, and bloody evacuations and tenesmus.

The causative bacteria may be organisms normally present in the intestinal tract which in the presence of an environment suitable to their abnormal growth, become pathogenic to the host, or the offending organism may be introduced from without through food or by contact with infected excreta or other matter.

The organisms which have been most frequently isolated from the intestinal tract during the more recent epidemics are the B. paratyphosus group, of which the alpha and beta types are probably the most frequent of-

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[1] Smillie: Am. Jour. Dis. Child., 1917, xiii, 337.

fenders in paratyphoid fever in the young. Both of these organisms are occasionally found in the intestinal contents and feces of children who give no history of infection.

The various types of B. dysenteriæ have been isolated in numerous severe epidemics, in both infants and children.

While this group covers a considerable number of bacilli showing minor morphological and cultural differences, they may, however, be divided into two main groups, the division being based on the difference in their reaction with litmus mannite. The first group is known as the "true Shiga," or "alkaline type"; the second as the "acid type," which includes the organisms which have most frequently been found in the diarrheal diseases of infants and children in the United States. This second group includes the Flexner-Harris, the "Y" type of Hiss and Russell, and the Strong subvarieties. The "Shiga" type has been found in some cases alone and even less frequently it has been isolated in cases in which the "acid" types were present. Both of these latter findings have, however, been the exception in the dysenteries of children.

B. typhosus, while not an infrequent cause of severe infectious diarrhea in infancy has only rarely been identified as the specific organism in generalized epidemics.

B. coli may become so abundant and virulent as to produce severe types of enteritis. Streptococci have been reported repeatedly as the predominating organism in isolated epidemics of severe types associated with deep intestinal lesions.

The rôle of B. aërogenes capsulatus (gas bacillus of Welch) as a specific factor in epidemics is still in dispute. It has been isolated in large numbers and was a constant finding in several epidemics. It has a profound influence in the reaction to diet in such cases and is re-

sponsible to a large degree for the character of the stools when present in large numbers.

B. pyocyaneus, B. lactis aërogenes and the proteus group as specific factors are open to question and it is more likely that they were accompanying rather than specific in reported epidemics.

Kendall and Day, making a careful study of the epidemics of summer diarrhea in Boston, found that during the year 1910 the epidemic was mainly due to dysentery bacilli, fully 75 per cent. of 52 cases being due to these organisms. Streptococci were also present in about 60 per cent. of the dysentery cases, probably as secondary or terminal invaders. The summer of 1911 was noteworthy as a "streptococcus" year; 54 per cent. of 146 cases studied harbored large numbers of these organisms. The year of 1912 was a "gas bacillus" year, these organisms appearing in unusually large numbers in 39 per cent. of 135 cases examined. Each of the above types was found each year, but the striking feature is the shifting of the dominant organism from year to year. Kendall concludes that, bacteriologically considered, these cases are of varied etiology, caused by organisms of very unlike characteristics.

In contrast to this, studies of the summer diarrheas at the Sarah Morris Hospital for Children (Chicago) during the course of two summers, showed that most of them were secondary to parenteral infections (see page 280). Day worked both in Boston and Chicago cases, and therefore the error could not have been one of technic. The difference was probably due to use of boiled milk in Chicago, and unboiled milk in the East. The infection in most cases probably being due to organisms ingested from the upper respiratory tract.

Pathology. To the invasion of pathogenic bacteria the digestive canal reacts by inflammation of the intestines (enteritis). The large intestine is always more affected, while in the small intestine the pathological

process, as a rule, is limited to its lower portion. However, in cases secondary to infections of the nose and throat, even the gastric mucosa may be involved. Mesenteric lymph-glands are swollen. In some cases the bacteria invade the deeper organs also, and may be cultivated from the spleen and the gall-bladder. Liver and kidneys show degenerative changes in severe cases, probably due to the action of toxins. Occasionally other organs may secondarily become affected (otitis, pneumonia).

The inflammation of the intestines may reach any degree of severity, and is dependent to some extent at least upon the causative organism, being, as a rule, most marked in cases in which dysentery, typhoid, and streptococcic organisms are excitants of the pathological process.

It may be a hyperemia and swelling associated with exudation of excessive amount of mucus and occasionally of blood, producing a picture of catarrhal gastroenteritis marked by mucus, mucopurulent, and occasionally also slightly bloody diarrheal stools. These cases are caused by a variety of bacteria, and they are often secondary to infections of the respiratory tract, the same micro-organisms being causative in both instances. We have frequently seen such a clinical picture associated with severe vomiting, and a secondary acidosis following in the course of a streptococcus sore throat.

Intense swelling of Peyer's patches in the small intestine is seen in typhoid infection. Sloughing and ulcer formation is far less frequent than in adults.

In paratyphoid infections, while infiltration of Peyer's patches and solitary follicles are usually present, deep ulceration is lacking, as a rule.

In infection with dysentery bacilli, the large intestine is especially affected, being the seat of sero-hemorrhagic and hemorrhagico-purulent inflammation, with marked

tendency to formation of ulcers throughout a large part of the large intestine, and less frequently in the course of the ileum.

Again, we may see marked intestinal pathology, as evidenced by deep-seated ulcerations and infiltrations of mucosa and secondary inflammation of the submucous and muscular layer of the intestinal wall, which condition is usually spoken of as ulcerative follicular colitis, and this may be complicated by formation of a pseudomembrane in various areas throughout the large intestine, which condition has been described as a membranous colitis. In many of these cases it is difficult to determine the exact bacteriological factor, because of the presence of secondary organisms. Most of these cases are either subacute or seen as secondary involvement in infants who have suffered from repeated nutritional disturbances.

On the whole, in those cases of inflammation of the intestinal tract due to bacterial infection and presenting serious pathological changes, the most marked changes are found in the lower three feet of the small intestine and in the large intestine. While there is very frequently a disparity between the severity of the clinical symptoms and the pathological changes seen *post-mortem* in that not infrequently severe symptoms are associated with little pathology, on the other hand marked pathological changes are almost invariably associated with a severe clinical picture.

Symptoms. The symptoms vary with the individual excitant of the disease, and thus also to a certain extent with the pathology, but, in general, the symptoms are so variable and with very few exceptions so little characteristic for the particular excitant that the etiological and pathological grouping of clinical pictures is impractical. It seems much better to differentiate the various forms from the clinical point of view.

Diarrhea with slimy or purulent evacuations, often with blood, accompanied by abdominal pain, tenesmus

and fever, are the most characteristic and the most constant symptoms of enteral infections.

The *onset* and *progress* of enteral infections, as a rule, are sudden and rapid, and in this way they markedly differ from alimentary nutritional disturbances in which prodromes consisting of milder symptoms are often present, and the progress is gradual. In enteral infections the stormy course may result in rapid production of a very severe picture of general prostration, and even an early fatal outcome.

Diarrhea is so constant that these cases have been designated as "infectious diarrhea," and yet it should be remembered that typhoid and paratyphoid infections in young individuals may be associated with any degree of constipation early in the disease. The stools are, as a rule, frequent, often one every hour, and there are also cases in which the bowels seem to move almost continuously. The number of stools varies also, according to the seat of the most severe inflammation, and they are more numerous when the large intestine is chiefly affected.

Loss of weight, often sudden and marked, is always present, and is due to many evacuations, and also to accompanying nutritional disturbance.

Stools. The macroscopical appearance of individual stools varies not only with the etiological factor, but is also dependent to a great extent upon the reaction to food, and upon the intestinal pathology, and is therefore of little value in the etiological diagnosis of enteral infections. The size of the stools is indirectly proportional to their number. In the beginning they appear to be of normal composition, but sooner or later they are composed chiefly of mucus and blood, and occasionally pus may be seen, even by the unaided eye. Portions of the intestinal mucous membrane are seen in severe cases at the time of sloughing and ulceration. The odor of the stool varies with its composition, and thus with the

progress of the disease. In the beginning the odor is that of the normal stool; later stools, composed of mucus and blood, are almost odorless; and those containing large quantities of sloughs have often a putrefactive odor. The reaction of the stools varies also with their composition, being mostly alkaline. In exceptional cases the stools may not be considerably increased in number, and may contain neither mucus, nor blood, nor pus.

Abdominal pain and *tenesmus,* due to irritation by the bacteria and their products, and also due to the abnormal intestinal contents, and to increased peristalsis, and sometimes to distention, appear very early in the disease, often being the first symptoms. Although being severe usually, they vary from a slight discomfort to excruciating pain, which keeps the child constantly awake, and, causing exhaustion, adds to the severity of the case. Abdominal distention is intermittent, the abdomen being usually sunken. Abdominal tenderness is not frequent. Anorexia is almost always present, while vomiting is more commonly seen early.

Fever is always present in enteral infections, and varies with the severity of the infection and the pathology. More often it is not extreme after the first exacerbations. It persists·throughout the disease.

Leucocytosis and oliguria are usually present.

Enteral infections are always associated with *nutritional disturbances,* since the infection affects an organ chiefly concerned in nutritional processes. And nutritional disturbances, again, produce symptoms of their own.

The *course* of enteral infections varies considerably, being dependent chiefly upon the nature of the organism and the stage of nutritional disturbance that develops, and also on the nature of complications. Some cases may be so mild as to resemble subacute dyspepsia, and only inability to influence the fever by the diet may point to their true nature. On the other hand, however, severe

toxic conditions occur, being due either to sepsis or to a nutritional disturbance which develops secondarily to infection. The duration of the disease varies from a few days to several weeks.

Complications. The great danger of the infections of the gastro-intestinal tract lies in their tendency to complications, at the head of which stand nephritis and pneumonia. Other complications are cysto-pyelitis and various pyodermatoses, and other pus infections and general pyemia or septicemia, which start either from the skin or from the diseased intestines.

More important than this is the association of infectious diseases of the intestines with secondary nutritional disturbances. It is easy to understand that in severely diseased intestines the normal digestion of food is made especially difficult, and thus acid decomposition easily occurs, which in turn leads to dyspepsia, and in the wake of these even the alimentary decomposition and alimentary intoxication may be implanted upon the original disease. The inanition caused by the too prolonged feeding with an unbalanced diet, such as cereal waters (flour injuries, Mehlnährschaden) may in some cases reach disastrous gravity. There can be no doubt that the majority of the cases resulting in athrepsia are not due to the infection alone, but also to the inanition and other forms of secondary nutritional disturbances, and it is probable that even a part of the severe ulcerative forms and various complications develop on the same foundation. The underfeeding alone gradually decreases the general power of resistance of the body; it weakens also the antibacterial functions, and thus the local or general infection may spread unimpeded.

Diagnosis. In making a diagnosis it is necessary to differentiate the enteral infections not only from (1) alimentary nutritional disturbances, but also from (2) nutritional disturbances caused by parenteral infections. (3) Diagnosis of the causative organism or group of

organisms is also of great importance for the treatment.
(4) Enteral infections are always complicated by nutritional disturbances, and it is of great importance to recognize the degree (simple infectious diarrhea, anhydremia and intoxication) to which the infant is affected.

In practice it is often difficult to differentiate clinically the gastro-intestinal infection from other forms of alimentary disturbances, because neither bloody and purulent stools nor the finding of pathogenic bacteria in the stools in itself is sufficient for the diagnosis of enteral infection, except possibly in the presence of typhoid, paratyphoid, and dysentery bacilli.

An easily applicable method of differentiation is the test for the *reaction to starvation and feeding*. Fever continuing after withdrawal of food speaks for infectious etiology. Inability to influence the symptoms by diet is to be interpreted in the same sense.

History is of considerable importance in making a differential diagnosis. The acute infectious diarrhea starts usually suddenly in a previously well baby, and prostrates it at once, while the alimentary nutritional disturbance comes on gradually. In the latter we get a history of improper feeding, of previous nutritional disturbance, of parenteral infection. It is more gradually progressive.

The differentiation between the enteral and the parenteral infections is somewhat more difficult, and is to be made chiefly by exclusion of the parenteral infection by careful physical examination of the patient. The bloody, purulent stools are usually absent in the cases secondary to parenteral infection.

The diagnosis of the causative organism is to be made by proper bacteriological examination and culture of the stools, and by agglutination reaction. Kendall states that frequently it is very difficult to determine the organism causing the disease, and therefore he has attempted to classify the causative organisms into two groups with a

While agglutination reactions are uncertain in very young infants, because of the slight tendency to the formation of agglutinins, in older infants and children they are of very considerable value, as demonstrated by the study of agglutinins by the author at Cook County and Sarah Morris Hospitals during the year 1914. In a series of 30 cases studied in which agglutinations were made for typhoid, paratyphoid (alpha, beta, and Morgan), dysentery (Shiga and Flexner), and colon bacilli, the following organisms were demonstrated: typhoid, 2; paratyphoid (Morgan), 1; dysentery, 2. All of these cases yielded the respective organisms in large numbers from their stool cultures. This method of examination is easily carried out in a well regulated laboratory, and is worthy of further consideration in the presence of an epidemic of enteritis or isolated cases of severe enteral infection.

The stage of the nutritional disturbance is best diagnosed by the reaction of the temperature and toxic symptoms to complete withdrawal of food, and presence or absence of paradoxical reaction. Starvation in the presence of infection must always be recognized as a dangerous procedure.

Prognosis. The prognosis of enteritis is, in general favorable. Death is almost always due to complications with septic affections or nutritional disturbances.

The mortality rate varies greatly in different epidemics. The same organism may in one year cause a widespread mild epidemic; at another it may be associated with a high mortality. Epidemics among institutional infants must always be given serious consideration.

In infants and younger children the prognosis depends essentially upon the ability of the physician to apply the proper dietetic methods suitable for the particular case. If he succeeds—and this is at present possible in very many cases—avoiding grave secondary nutritional disturbances, then he will be able to save a surprisingly

large percentage of cases; if he is unsuccessfu. in this direction, then his results will be unsatisfactory.

Treatment. Prophylaxis. In the etiology of enteral infections several facts based on bacteriological studies and clinical observations stand out so prominently that the methods of prophylaxis must be based upon them in order to be successful.

1. In the great majority of cases the infection is introduced with the food. Whenever intestinal infection occurs in a breast-fed infant in a private home, the first thought should be that the infant was probably getting other food besides mother's milk, and only after exclusion of this probability should the causes be looked for in the environment of the infant, especially the cleanliness of the mother and the general hygiene of the home. In artificially fed infants the prophylaxis of enteral infections consists of obtaining pure and wholesome milk, keeping it clean, boiling when in doubt, and careful preparation of proper mixtures.

2. In many cases the infection occurs by contact, especially in institutions. Isolation of severe cases of intestinal infection is therefore essential, and isolation of all suspicious cases advisable, especially in institutions.

3. The environment of the infant, and especially lack of proper cleanliness generally, and in preparation of foods especially, are very frequently predisposing and accessory factors. The methods instituted to counteract these influences must, of course, be adapted to the individual case.

4. Parenteral infections are often followed by enteral infections. Proper treatment of parenteral infection, special attention to the diet and general hygiene, are the keynote of prophylaxis in these cases, the possibility of secondary enteral infection being constantly kept in mind.

5. Alimentary nutritional disturbances increase susceptibility to any form of infection. and especially to enteral infection, and the prophylaxis of secondary enteral in-

fections coincides practically with the prevention and proper treatment of these nutritional disturbances. (See also "Susceptibility Influenced by Nutrition," p. 276.)

Initial. The object of the initial treatment is to decrease as much as possible the number of bacteria present in the intestine, and the removal of irritating intestinal contents. Intestinal disinfection by drugs is impossible; and the cleansing of the intestines by the administration of large quantities of inert fluids, enemata, and possibly an initial laxative, is the best that can be done in this direction.

Castor oil, which is usually taken plain without any difficulty by infants, in doses of 1 to 2 teaspoonfuls, is the best laxative for these cases, since it causes very little intestinal irritation. Only in cases where it is vomited, we should resort to magma magnesiæ ($\frac{1}{2}$ to 4 teaspoonfuls), or to calomel, 0.06 gram (1 gr.), given in doses of 0.015 gram ($\frac{1}{4}$ gr.) every half an hour until two to four doses are given. Calomel should always be administered with caution and is best used with sodium bicarbonate. The calomel can be followed to advantage with 1 or 2 teaspoonfuls of magma magnesiæ.

An enema of physiological saline (1 teaspoonful of salt to 1 pint of water) is useful.

All food should be stopped for from six to twelve hours. It is not desirable, as a rule, to withhold the food longer than this time.

Water should be given freely during the starvation period. The water may be given either warm or cool, or in the form of weak tea. Saccharin may be used to sweeten it, using 0.01 gram ($\frac{1}{6}$ gr.) of saccharin to 8 ounces of water, if desired. In presence of marked anorexia or refusal of fluid on the part of the infant, the water or tea must be administered by catheter. In persistent vomiting, frequent resort to gastric lavage with 1 per cent. sodium bicarbonate solution will relieve vomiting, and be followed by retention of fluids given by mouth.

When the latter does not relieve the vomiting, physiological saline solution or Ringer's solution must be given either by rectum or subcutaneously.

Local and Medicinal Treatment. *Abdominal pain* and *tenesmus* are often so severe that they require a special treatment. Moist heat in the form of compresses, hot water bottles or electric pads should be given preference, and only in cases in which they do not afford relief should recourse be had to opium or morphine. Tincture of opium in doses of 3 to 5 drops may be given in 10 per cent. starch solution by the rectum, or 0.01 to 0.03 gram ($\frac{1}{6}$ to $\frac{1}{2}$ gr.) of pulvis ipecacuanhæ et opii (Dover's powder) (beware of vomiting), or 5 to 20 drops of tinctura opii camphorata (paregoric) by mouth. In some cases 1 or 2 doses of morphine may be preferable, since it decreases the peristalsis less markedly than opium; the dangers of its administration to infants must be remembered, and the dosage must be minimal (0.0003 to 0.001 gram—$\frac{1}{200}$ to $\frac{1}{60}$ gr.). Atropin—$\frac{1}{1500}$ to $\frac{1}{500}$ gr. and epinephrin in 1:1000 solution, 1 to 5 minims, may be indicated in the presence of marked atony of the intestinal tract. Argyrol in 6 grain doses, administered as a 10 per cent. solution, may have a beneficial influence. (See page 236.)

Stimulants are indicated in some cases of extreme exhaustion, and in cases of sudden collapse. In the absence of hyperexcitability of the nervous system, strychnin is the most generally useful stimulant. It is given in doses of 0.00005 to 0.0003 gram ($\frac{1}{1000}$ to $\frac{1}{200}$ gr.). Caffein in the form of caffein sodium benzoate, or citrated caffein, are of value, and is given in doses of 0.01 to 0.03 gram ($\frac{1}{6}$ to $\frac{1}{2}$ gr.). Camphor 0.05 to 0.10 gram (1 to 2 gr.) dissolved in sterile oil may be injected subcutaneously in emergency, to be repeated as indicated.

Special symptoms and conditions arising during the course of the disease, as high fever, excessive vomiting, symptoms of nervous excitation, or extreme depres-

sion, are to be treated as detailed under Anhydremia page 259).

Injections of silver nitrate are of value in some cases where blood and pus persist in the stool even after the subsidence of acute symptoms, and especially in dysentery. Before an injection is given, the colon should be irrigated first with sterile water (not saline). One per cent. silver nitrate solution is then injected in a suitable quantity. If it causes any pain or irritation, it should be washed out with saline solution. It should not be repeated more often than once a day, and if three injections do not result in marked improvement it is better to discontinue them.

Dietetic Treatment. *Human Milk.* The ideal treatment for all cases of intestinal infections is by feeding with human milk, and whenever obtainable, more especially in the severe types it is by all means the diet of choice. Feeding with human milk, especially in young infants, produces very good results, because it retards the complicating nutritional disturbance, and thus favors healing.

Artificial Feeding. From the great number of food mixtures that have been advised for enteral infections, we may judge as to the difficulty of feeding in such cases. It is probable that success may be obtained with any feeding which prevents the aggravation of nutritional disturbance, and favorably influences the nutritional disturbance which may exist. Feeding with albumin milk, skim and buttermilk, and cereal mixtures and whey-cereal mixture (Frank) offer the least risk.

Prolonged starvation by insufficient diet or by refusal on the part of the infant to take the prescribed diet is always disastrous, and must be avoided. After six, or at the most twelve, hours on the tea diet the infant is placed on cereal water (barley, rice, or flour ball), using 1 tablespoonful of the flour to a pint of water in young infants, and 2 tablespoonfuls to the pint of water in

infants over 1 year. After twelve to twenty-four hours on the above diet an ounce of young chicken, veal or lamb broth can be added to the above cereal waters, seasoning with a small amount of salt. If the child will take the food, it may be given in the same quantities to which the child has been accustomed, or smaller quantities at more frequent intervals.

By far the best results obtained in our private and hospital work have been by instituting feeding with the albumin milk[1] of Finkelstein after the first twenty-four hours on an inert diet. The value of the albumin milk may be explained by the fact that it is easily digestible, containing moderate quantities of fat and sugar and finely divided casein, which is easily digested in this form. The rules to be followed in the feeding with albumin milk are described under Diarrheal Disturbances. (Page 234.) This diet is also to be recommended in home practice, wherever it is possible to obtain it, either from a neighboring hospital or by instruction of the nurse or of the mother. Feeding with albumin milk should be begun after twenty-four hours on the tea and cereal water diet. Sufficient quantity of inert fluid, either in the form of water, tea, or cereal water should be given with or between the small feedings of albumin milk. One of the gravest dangers in the severe infections is that the infants are likely to take too little rather than too large quantities, and are especially prone to vomit when the food is forced upon them.

Boiled skim buttermilk or skim milk with starch or flour ball added (1 tablespoonful to the pint) may be used as substitute, if albumin milk cannot be obtained. They are, however, not so efficacious. They should be fed in small quantities, as recommended for albumin milk.

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[1] Dilutions of the dry forms in which it is obtainable on the market will answer. (See Appendix.)

Chymogen milk (either made from the whole milk, or in severe types from skim milk), either diluted or in small quantities, if given full strength, is frequently retained when the stomach is very irritable, and where the child objects to the less palatable albumin milk and buttermilk mixtures.

For further treatment see Diarrheal Disturbances.

"The whey-cereal mixture therapy of Frank deserves mention. It is administered as follows:

1st day: Initial starvation period on tea for not longer than twelve hours.

2d day: Feed five times 50 grams whey and 50 grams cereal gruel prepared from crushed grain.

3d day: Increase to 60 grams whey and 60 grams cereal gruel.

4th day: 75 grams whey and 75 grams gruel.

5th to 8th day: Not later than on the fifth to eighth day of treatment replace a tablespoonful of whey by tablespoonful of milk. Increases of milk to be guided by the infant's progress and needs.

9th to 11th day: Increase the addition of milk gradually.

12th to 14th day: Even in the grave case 400 grams of milk and 400 grams of cereal gruels and 200 grams of meat broth must be given, and not later than in this time the broth is to be prepared with strained rice or farina. In infants over 1 year, beginning with the tenth day, finely scraped beef may be added."

A careful record should be kept of the exact amount of milk and other fluids taken in each twenty-four hours, and, where possible, the child should be weighed daily to ascertain the loss in weight.

The dietetic therapy has never such a prompt result as in alimentary nutritional disturbances. Even in favorable cases the disease (purulent and bloody stools, fever) continues for one week; in unfavorable cases, several weeks. Strict adherence to the food régime once insti-

tuted is desirable. In these cases no greater mistake could be made than to change diet with introduction of repeated hunger days, or to allow the infant to remain on small quantities of food. Thus, an infant suffering from infection succumbs often not to the infection, nor to the nutritional disturbance, but to inanition.

Diet in Convalescence. The problem of nutrition offers great difficulties, even after the subsidence of the fever, and following the improvement in the number and character of the stools, as it is frequently necessary to keep the infant on a restricted diet for from one to three weeks. Only rarely it is possible to feed sufficient caloric units for the maintenance of weight during the first and the second weeks of the illness. Where possible, the albumin milk, buttermilk, skim milk, and chymogen milk and cereal gruels should be gradually increased, and these increases in quantity should be maintained even in the presence of moderately bad stools if vomiting is absent, unless one becomes convinced that one or the other of the food elements is absolutely detrimental to the infant's welfare.

It is our desire to impress that possibly the gravest danger to the infant during the period of convalescence is that of underfeeding. Upon the return to milk mixture small quantities of boiled milk, low in fat (albumin milk, buttermilk, skim milk) should at first be used. This may be accomplished by adding it to the cereal gruels. During this stage beef juice broths, egg albumin, coddled egg (prepared as for typhoid fever patients), zwieback crumbs, pap, custards, and junket may be added. Under conditions where ideal milk and milk preparations cannot be obtained, we have found that not infrequently the better brands of evaporated milk, as obtained on the open market, are useful, when properly diluted. The use of condensed milk should be avoided.

The obstinate constipation which is sometimes seen during convalescence should be treated with the utmost

conservatism along the lines as laid down for constipation. The infant should have at least one evacuation of the bowels daily. A saline enema is usually sufficient to produce this result.

PART V.

Rickets (Rachitis).

RICKETS is a chronic general disorder of nutrition. The predisposing etiological factors have been, to a considerable degree, clarified by recent investigations and it is to be expected that from the clinical and technical studies now in progress, considerable new knowledge as to the exact etiology will be forthcoming in the near future.

The pathological changes, while most evident in the osseous structure, may involve all of the body tissues and organs, including the nervous, muscular, respiratory and circulatory systems, the blood-making organs, liver, skin, hair, teeth and nails.

Its clinical manifestations are most commonly evidenced in the second half of the first year in the full term infant. In the premature and congenitally diseased infants, and not infrequently in other infants, it can be diagnosed even before the third month of life. Radiographic diagnosis is usually possible before clinical signs are sufficiently manifest to warrant a conclusive diagnosis. However, in the first stages they are frequently overlooked because of the permeability of the rarified structure to the roentgen rays. The bones may be but slightly increased in density over the surrounding soft tissues. The diagnosis is usually made upon the pathognomonic bone lesions of the second and third stages. The bone lesions of the first stage are less frequently recognized but are of even greater importance for the adoption of proper hygienic, dietetic, and medicinal therapeutics for the prevention of permanent tissue changes

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and deformities. The treatment demands a general knowledge of all of the possible underlying etiological factors, the associated general systemic as well as the bone changes and the possible progressive bone deformities which may develop even after the predisposing factors have been removed and the acute process arrested.

Etiology. Most of the theories as to the exciting causes can be grouped into four classes, namely: *dietetic, hygienic, infectious,* and *endocrinous.*

While this disease is in all probability primarily a dietetic disorder, in most cases in the human, improper hygienic conditions and infections may hasten the development of the pathognomonic bone and systemic changes and precipitate the clinical manifestations, more especially of the secondary complications.

Rickets is the most common chronic disorder of infants living in the Temperate Zone. Schmorl,[1] in a series of histologic studies, found that 90 per cent. of the children in his Dresden clinic, dying before the age of 4 years and examined at autopsy, had rickets, either manifest or latent. Dick[2] found that 80 per cent. of the children examined in the London county schools had the disease or had suffered from it previously. Kassowitz[3] calculated that 90 per cent. of the children in the out-patient department of his Vienna clinic were rachitic. Epstein[4] gives about the same figures for Prague, and Kissel[5] for Moscow. Engel[6] states that in Germany, during the late war, all young children were affected to some degree. He attributes this especially to the lack of fresh milk and fats during the war.

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[1] Schmorl: Ergebnisse der Inneren Medizin u. Kinderheilkunde, 1909, p. 403.

[2] Dick, L.: Proc. of Roy. Soc. Med., 1915—Brit. Jour. Dis. of Children, 13—332, Nov. 16.

[3] Kassowitz: Modern Clinical Medicine—Dis. of Children, Julius Springer, Berlin, 1910, p. 239.

[4, 5] Epstein and Kissel: *Ibid*, p. 239.

[6] Engel: Lancet 1, 188, Jan. 24, 1920.

E. J. Huenekens[1] reports that 39 per cent. of the infants in his Minneapolis Welfare Clinic showed definite clinical signs of the disease. Hess[2] reports 90 per cent. of the infants in a New York City infants' asylum as showing evidence of rickets.

It must be remembered that the figures above quoted are taken from patients among the poorer classes. Abroad and in this country out-patients are also composed largely of a class, many of whom would be subjects of improper artificial feeding or as disastrous underfeeding, or improperly balanced diet and overwork in the lactating mother. These figures must be necessarily high when compared with those of the better classes, and more especially children from rural districts, who live in the open and receive an abundance of fresh milk and green vegetables.

Interesting and important is the recognition of the effect on the development of rickets in the offspring due to the changes of *climate, environment* and *diet* in the parent.

In the United States almost all the negro infants in northern cities have rickets, whether bottle- or breast-fed, although it is more frequently of a severer type in the artificially fed. It is far less frequent in negroes of the southern states and uncommon in negroes of Cuba and the West Indies.[3] The condition is also very prevalent among Italians in the United States, although infrequently seen in the rural districts of Italy. It occurs more commonly and severely among the poorer of these individuals.

Hess and Unger found that the diets of lactating northern negro mothers were deficient in fresh vegetables and fruit, and contained an excess of carbohydrates, the diets, therefore, being poor in calcium, phos-

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[1] Huenekens, E. J.: Jour. Lancet, 37, 804, Dec. 15, 1917.
[2] Hess, A.: Jour. Am. Med. Assoc., 76—698, March 12, 1921.
[3] Hess and Unger: Jour. Am. Med. Assoc., 70—900, 1918.

phate and the vitamines. It must be remembered that the human body in all probability cannot synthesize the vitamines but must depend upon the amount eaten for that to be contained in breast milk. The mother's diet thereby directly influences the amount fed to the sucklings. The same factors, in all probability, play an important rôle among the Italian immigrants. Both of these classes of individuals also show a tendency to nurse their offspring beyond the point of their physical and physiological capacity.

Neve[1] called attention to the fact that rickets is exceedingly rare in parts of India where infants live under the worst conditions of hygiene and diet, but where sunlight, to which they are almost constantly exposed, abounds. Similar observations have been made in the Hebrides, off the coast of Scotland, where many infants live in dark, damp houses during their first year, but in whom rickets is rare, due to the fact that the mothers live largely on fish, especially codfish livers, and milk products and moderate amounts of vegetables. On the mainland of Scotland, where the diet of the mothers is largely made up of milled cereals, muscle meat, and potatoes, rickets is very prevalent although the hygienic surroundings are much better.

Season. It is more active in the winter and spring months and in this peculiarity, hygiene, lack of exposure to sunlight, insufficiency of fresh foods in the mother's or cow's diets, and intercurrent infections may one and all be contributory factors.

The *age* of onset varies considerably. In the mature infant the symptoms usually manifest themselves between the sixth and eighteenth months. Some observers describe as congenital rickets, a condition involving the osseous system which develops intra-uterine, presenting similar pathological findings at birth. There has been a great deal of contention as to the exact nature of these

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[1] Neve, E. F.: Brit. M. J., Vol. 1, 518, 1919.

cases; they undoubtedly, however, belong to the osteo-genesis imperfecta or osteopathyrosis group.

Heredity has been designated as a predisposing factor in exceptional cases.

Prematurity is a predisposing factor to the development of the disease and the more prematurely born and consequently the more poorly the physiological functions are developed, the greater likelihood is there that the infant will suffer from rickets. Huenekens[1] found 92 per cent. of his premature and twin pregnancies showed rickets in his series of sixty cases. He reports that 81 per cent. developed clinical evidence of rickets between the third and fourth month of life. Many of my prematures developed clinically evident rickets before the end of their second month. This frequency and early development may be due to the fact that the mineral content of prematures is below normal, as two-thirds of the minerals are deposited during the last few months of fetal life, the fetus at six months containing thirty grams and at nine months one hundred grams.—Birk.[2] In the newborn, fully 75 per cent. of the ash content consists of calcium and phosphate, consequently rickets being a disturbance in the calcium and phosphate metabolism, the more premature the infant, the greater the deficiency in calcium and phosphorus, so that the surplus supply is exhausted earlier than in the full term.

Underfeeding is another factor in the development of rickets in the premature. The low calcium content of human milk and the difficulty of metabolizing even this food in sufficient quantities to prevent drawing on the inherited supply may be an active factor. Huenekens[1] believes that this condition in the premature may be a pseudo-rickets because it is based upon a deficiency in calcium rather than a disturbed calcium metabolism as in true rickets.

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[1] Huenekens: Jour. Lancet, xxxvii, 804, Dec. 15, 1917.
[2] Birk and Orgler: Monatschr. f. Kinderh. 1, 1910, 544.

Diet. The artificially fed are especially prone to develop severe rickets. If the diet contains sufficient milk, the tendency to develop the disease is less than when fed mainly on cereals and proprietary cereal foods with only small amounts of milk.

McCollum, Shipley and their co-workers,[1] in a study of the effect on the growth and development in rats by the feeding of more than 300 different diets came to the conclusion that the etiological factor is to be found in an improper dietetic regimen. They were able to produce approximately normal nutrition and normal skeletal growth when properly constructed diets were fed. They were able to develop pathological conditions in rats corresponding in all fundamental respects to rickets in humans through dietetic insufficiencies: (1) By diminishing the phosphorus and supplying the calcium in optimal quantities or in excess, or (2) by reducing the calcium and maintaining the phosphorus at a concentration somewhere near the optimal. They believe that rickets is essentially an expression on the part of the skeleton of disturbed relations between the calcium and phosphate ions of the body fluids and that there are two main kinds of rickets: one characterized by a normal or nearly normal blood calcium and a low blood phosphorus (low phosphorus rickets); the other by a normal or nearly normal blood phosphorus but a low blood calcium (low calcium rickets).

Due to the fact that when they fed diets containing a complete salt mixture, the rats did not develop rickets, even when there was an absence of fat-soluble vitamine in the diet, they concluded that a deficiency in this substance cannot be the sole cause of rickets. They,

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[1] McCollum, Simmonds, Parsons, Shipley: Jour. Biol. Chem., Vol. 45, 333, 1921. Shipley, Park, McCollum, Simmonds: John Hopkins Hosp. Bull., Vol. 32, 160, 1921. McCollum, Simmonds, Shipley, Park: Jour. Biol. Chem., Vol. 50, 5, 1921. Shipley, Park, McCollum, Simmonds: Am. Jour. Dis. of Children, Vol. 23, 91, 1922.

however, feel that these findings should not exclude the
fat-soluble substances from consideration as etiological
factors in the production of rickets and kindred diseases,
believing that there is a possibility that the blood phos-
phate and calcium level may be determined in part by
the amount of fat-soluble substances available for the
needs of the organism.

They pursued investigations with a view to determin-
ing the nature of the substances contained in certain
animal fats which protect the skeleton when the calcium
content of the diet is unfavorable to the formation of
normal bone. In this series of observations the phos-
phorus content was in every case not far from the
optimal. They were led to the conclusion that codliver
oil contains in abundance some substance which is pres-
ent in butter fat in but very slight amounts and which
exerts a direct influence on the bone development.
They further believe that this substance is apparently
distinct from fat-soluble A.

They made further studies on the effect of sunlight
and ultra-violet rays on bone development. These ex-
periments led them to suppose that in the absence of
certain active light rays any influence which would re-
sult in the depression of the calcium or phosphate ions
in the body fluids with the formation of calcium-phos-
phate ratios favorable for the development of rickets
would ultimately produce the disease.

Mellanby,[1] experimenting on puppies, was led to be-
lieve that rickets was dependent upon calcium, phos-
phorus, and fat-soluble vitamine deficiency, stating that
foods which were rich in these substances prevented
rickets, while those poor in them apparently caused it.
He also believes that exercise is important in the pre-
vention of rickets.

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1 Mellanby. E.: Experimental Rickets, Med. Research Council,
London, 1921.

Hess and Unger[1] carried out a series of experiments on the relation of fat-soluble A to rickets, but were unable to confirm his findings. They studied institutional infants living under excellent hygienic conditions with good nursing and care, and food adequate in caloric content and containing sufficient anti-scorbutic. Those fed large amounts of milk (24 to 32 ounces of raw or pasteurized milk) developed rickets just as easily and to the same degree as the infants receiving only $\frac{2}{10}$ per cent. milk fat. Those on low fat showed no greater signs of rickets than the average baby in the institution, even though at the most vulnerable age. The babies fed large amounts showed no symptoms of indigestion which might have resulted in an inability to properly metabolize their diet. They conclude that the fat-soluble vitamine is not the much sought for anti-rachitic factor and that babies can thrive for a long period on a very limited amount of fat, providing that the diet is otherwise adequate. They feel that the infants must have utilized enough fat-soluble A vitamine from this large amount of milk fat, even if the vitamine content of the milk might have been small because of possible deficient diet of the cows. They state that it is their belief that rickets is primarily a dietetic disorder but that hygienic factors, such as sunlight, poor ventilation, and crowded quarters and infection, are important contributory agencies.

It is a common clinical experience to find that diets containing a minimum of fats and salts, composed largely of carbohydrates, which usually means cereals, result in the development of rickets. This poorly balanced combination is strikingly seen in the feeding with proprietary infant foods, to which a minimum of milk is to be added. A sweetened condensed milk diet offers a good example of what may be expected from overheating, ageing, and a low fat and high sugar feeding.

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[1] Hess and Unger: Jour. A. M. A., lxxiv, 217, Jan. 24, 1920.

The theory of *defective hygienic* conditions and do-
mestication, particularly in crowded cities, was first ex-
pounded by von Hansemann.[1] Findlay[2] believes that
poor housing conditions, lack of fresh air and exercise,
are important factors.

Hess and Unger[3] were the first to demonstrate by
means of the roentgenograph that sunlight alone exerts
a curative action in rickets. They also pointed out the
possible rôle of actinic rays in an interpretation of the
seasonal variation in the disease and expressed the
opinion that it is the dominant factor in this incidence.
They do not imply the diet is not of importance in the
etiology of rickets, but rather that a hygienic factor—
sunlight—also needs to be taken into account. They
also confirmed the work of Huldschinsky and others,[4]
to the effect that the ultra-violet rays from a quartz lamp
and other sources had a similar curative value. Powers
and his co-workers[5] demonstrated the effect of the ac-
tion of light in rickets in rats by a series of experiments.
The diet used was the same in all animals and contained
an optimal content of calcium and was decidedly below
the optimum in its content of phosphorus and fat-soluble
A, and in other respects it was well balanced. The
experiments covered a period of two months. The ani-
mals which were kept in sunlight with a daily average
of four hours of exposure, did not develop rickets and
became sexually active. The control animals, which were
kept in an ordinary laboratory, showed all the gross and
microscopic evidence of rickets. In the animals exposed
to sunlight on this poorly balanced diet, while not show-
ing rachitic changes in the bones, there resulted more

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[1] Von Hansemann : Berliner klin. Woch., 1906, 20—21.
[2] Findlay : Brit. Med. Jour. 13, July 4, 1908.
         Glasgow Med. Jour., 89, 268, May, 1918.
         Med. Res. Committee Report, 20, 1918.
[3] Hess and Unger : Jour. A. M. A., 77, 39, 1921.
[4] Huldschinsky : Deutsch. Med. Wochnschr., 45, 712, 1919.
[5] Powers, Park, Shipley, McCollum and Simmonds : Jour. A.
M. A., 78, 159, 1922.

delicate bone structure than in animals placed on a properly balanced diet under the same conditions; showing that while the sunshine completely prevented the development of rickets it did not entirely compensate for the deficiency of phosphorus in the diet, as regards the growth and development of the rat as a whole, or of the skeleton, but did enable the organism to put into operation regulatory mechanisms which otherwise would have been inoperative or ineffective.

The *infection theory* as a direct cause of rickets has few followers except insofar as infection may interfere with metabolism.

The *endocrine gland theory.* The thyroid, parathyroid, thymus, and adrenal abnormalities have each in turn been described as being closely related to the development of rickets, but in all probability the dysfunction described has been secondary to the same factors causing the pathological changes in other tissues.

From the accumulated data on animal experiments and clinical studies the conclusions must be drawn that the *dietetic* and *hygienic theories* offer the best solution of the pathogenesis of rickets.

The question of race may predispose its development, as may heredity in exceptional cases.

The seasonal incidence is probably influenced by the confinement and poor hygiene during the winter and spring months and the frequency of infections during this period of the year. In the artificially fed, the winter feed of the cows may be an element.

Chronic constitutional and metabolic disturbances may act as contributory causes.

Mineral metabolism. Schabad and others have shown that the calcium and phosphorus balance is disturbed in rickets, the excretion of both phosphorus and calcium being increased in the acute stage and decreased during convalescence. It is certain that the calcium and phos-

phorus content of rachitic bone is much reduced from the normal.

The following facts are known regarding the metabolism of calcium and phosphorus: There is practically always a sufficient amount of calcium in the infant's food to meet its requirements for health and growth, although a calcium dietary deficiency may be present in the exclusively breast-fed. Breast milk contains 2 Gm. of ash per 1000 mils and 0.458 Gm. of calcium per 1000 mils. Cow's milk contains 7.5 Gm. of ash per 1000 mils and 1.72 Gm. of calcium per 1000 mils; thus the calcium content of cow's milk is four times that of human milk. The calcium in the milk is in organic combination although inorganic calcium may also be absorbed by the body.[1]

In Holt's[2] series of studies on calcium metabolism, healthy infants taking cow's milk modifications absorbed 0.09 Gm. of calcium oxide per kilogram of body weight, while breast-fed infants absorbed 0.06 Gm. per kilogram. Somewhat older children, taking a mixed diet, absorbed 0.055 Gm. per kilogram. All of these cases received a sufficient calcium intake. He further states that the intake of calcium oxide should be at least 0.19 Gm. per kilogram to insure the average absorption of 0.09 Gm. per kilogram in the artifically fed and at least 0.13 per kilogram to insure an absorption of 0.06 Gm. per kilogram, the amount absorbed by the breast-fed. In general he found that 35 to 55 per cent. of the calcium intake was absorbed. An excessive calcium intake did not increase the calcium absorption as the excess was excreted. If the intake of calcium oxide were very low, less than 0.10 Gm. per kilogram, the absorption of the

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[1] Aschenheim and Kaumheimer: Monatsch. f. Kinderh. X. 1911—12, 435.

[2] Holt, L. E., Courtney, A. M., and Fales, H.: Amer. Jour. Dis. of Children, Vol. 19, P. 97, Feb. 1920 and p. 201, March, 1920.

CaO was below the necessary requirements, and a negative calcium balance might develop. They found the best absorption of calcium obtained when the calcium intake bore a definite relation to fat intake. McCollum believes that in infants the reason that rickets develop is because the dilution of milk is such as to destroy or alter the proper mineral proportion when nothing else is added to the diet.

Excretion of calcium is almost entirely through the intestines, especially the large bowel, only 5 to 10 per cent. being excreted by the kidneys.[1] Children suffering from chronic intestinal indigestion show a low calcium absorption.

A diminished calcium retention (negative calcium balance) exists in the florid stage of rickets,[2] even though the intake is ample. The calcium retention returns to normal when the disease is cured, but during recovery. the absorption is higher and the excretion in the stools less than normal.[3] Howland and Park[4] have demonstrated a beginning calcium deposit in the bones of animals two days after beginning the administration of codliver oil. By the end of the third week after beginning the administration of codliver oil, calcium deposits can be demonstrated in the cartilages of human beings by means of the roentgenogram.

In prematures there is very low calcium retention during the first month of life, according to Hamilton.[5] This is probably dependent on several factors, among the most important being the low mineral content of prematurely born infants, and the restricted intake of foods with low calcium and phosphorus content, as is found

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[1] Voit, E.: Zeitschr. f. Biol., 1880, xvi, 55.

[2] Schabad: Arch. f. Kinderh., 1910, liii, 380.

[3]Holt, L. E., Courtney, A. M. and Fales, H.: Am. Jour. Dis. of Children, Vol. 19, p. 97, Feb., 1920, p. 201, March, 1920.

[4] Report of the 32d Meeting of Amer. Ped. Soc. Arch of Ped. xxxvii, 411, July, 1920.

[5] Hamilton: Am. Jour. Dis. of Children, Vol. 20, 316, 1920.

in breast milk and cow's milk in high dilutions. These infants almost invariably suffer from severe secondary anemias, dependent upon the same causes. Both rickets and secondary anemia are frequently prominent in this class of infants by the end of the second month of life. Rachitic premature infants also show bone changes which present a different pathological picture from the later rickets seen in full term infants, in that the skull is involved out of proportion to the clinical findings in the extremities and thorax. The result is a megacephalus characterized by a large, round head with prominent frontal and parietal eminences and open sutures, while the changes in the ends of the long bones and costochondral junctions show only a moderate degree of involvement.

The average normal blood calcium content for infants under 1 year of age is 10 to 11 mg. per 100 mils of serum.[1] Kramer and Howland[2] have demonstrated that the calcium concentration in the serum in normal children is singularly constant, so that even small deviations are regarded as diagnostic of disorder.

As previously stated, dependent upon the blood picture, there are in all probability two main kinds of rickets. One is characterized by a normal or nearly normal blood calcium and a low blood phosphorus; the other by a normal or nearly normal blood phosphorus but a low blood calcium. The investigations of Howland and Kramer,[3] and of Kramer, Tisdall and Howland,[4] on the calcium and phosphorus content of the blood serum in rickets and tetany have given suggestive evidence in support of this idea. These observers found that in children suffering from rickets alone, the phos-

[1] Brown, A.: Am. Jour. Dis. of Children, Vol. 19, 413, 1920.
[2] Kramer, B., and Howland, J.: Jour. Biol. Chem., xliii, 35, 1920.
[3] Howland, J., and Kramer, B.: Am. J. Dis. of Children, 22, 105, 1921.
[4] Kramer, B., Tisdall, F., and Howland, J.: Am. J. Dis. of Children, 22, 431, 1921.

phorus of the blood serum is low, and the calcium not far removed from the normal; in chîldren suffering from manifest tetany complicating rickets, on the other hand, the calcium is low but the phosphorus often not far removed from normal.

Phosphorus Metabolism. Phosphorus is contained in milk in the following forms: inorganic calcium phosphate, and as organic in combination with casein, nuclein, lecithin, etc. There is 0.294 to 0.418 Gm. per 1000 mils in human milk, of which 43.3 per cent. is organic, and there is an average of 2.437 Gm. per 1000 mils in cow's milk, of which 46 per cent. is organic.

Schabad[1] states that two-thirds more of the phosphorus is normally excreted by the kidneys, the rest from the bowel, the proportion being 80:20 in the breast-fed and 60:40 in the artificially fed. During florid rickets the relations are 65:35 and 40:60 respectively, and there is an absolute increased excretion of phosphorus. As the disease is arrested the proper proportions are re-established and for a time there is a greater retention of phosphorus than normal. Howland and Kramer[2] have shown that the blood phosphate is low in the blood plasma of rachitic children, and that the administration of codliver oil causes a marked rise. Hess and Gutman[3] have demonstrated similar changes following exposure to the sun's rays and by use of quartz lamps.

The phosphorus of the blood can be increased by feeding phosphorus per mouth. Marriott,[4] worked with artificial blood, found that by small increases in the phosphorus content, a precipitate resembling in composition the salts of bone was formed. Wegner[5] found that feed-

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[1] Schabad: Arch. f. Kinderh., 1910, liv, 83.

[2] Howland and Kramer: John Hopkins Hospital Bulletin, May, 1921, p. 165.

[3] Hess, A. F., and Gutman, P.: Proc. Society Exper. Biol. and Med., 19, 31, 1921.

[4] Report of 32d. Meeting of Am. Ped. Soc., Arch. of Ped., xxxvii, July, 1920.

[5] Wegner: Virchow's Arch. f. Path. Anat., 1872, 55, 9.

ing small doses of phosphorus to young, growing dogs and cats, caused an increased new bone formation, especially in the diaphysis along the epiphyseal lines. Adult bones were not affected. Kassowitz[1] confirmed the above work but noticed that feeding too large a dose of phosphorus caused osteoporosis. Phemister[2] applied these experiments to children and noticed by roentgen ray studies that phosphorus affected the normal bones of children as it did Wegner's animals and that the accumulation of calcium and overproduction of bone in the metaphysis continued for some time, even after the administration of phosphorus was discontinued. He[3] has more recently reported similar results in rachitic infants.

Further Relation of Diet to Calcium Metabolism. Howland[4] has demonstrated that carbohydrates in the diet favor a calcium retention. Freund[5] has shown that an excess of fat in the diet may cause a negative calcium balance. Holt found that the best absorption of calcium occurred when the food contained from 0.045 to 0.060 Gm. of calcium oxide for every gram of fat and the fat intake was ample, not less than 4.0 Gm. per kilogram. On a mixed diet a slightly lower proportion of calcium oxide to fat was needed to insure good absorption of calcium oxide. No constant relation between calcium and fat excretion was found. Lindberg,[6] however, demonstrated that in the breast-fed a high fat intake (breast milk enriched with added breast milk fat) may lead to at least temporary losses of calcium. This increased fat intake was followed by an increased fat

[1] Kassowitz : Ztschr. f. Klin. Med., 1884.

[2] Phemister, Effects of phosphorus on growing normal and diseased bones: Jour. Am. Med. Assn., 70—1737, June 8, 1918.

[3] Phemister, Effects of phosphorus on growing normal and diseased bones; Jour. Am. Med. Assn., 70—1737, June 8, 1918.

[4] Howland and Marriott: Am. J. Obstet., 1916, lxxiv, 541.

[5] Freund: Jahrb. f. Kinderh., 1905, lxi, 36.

[6] Linberg: Ztschr. f. Kinderh., xvi, 90, 1917.

excretion. Hamilton[1] suggests that the poor calcium retention in prematures may be related to the relatively large excretion of fat as their characteristic feces are rich in fats. Protein apparently has no influence on the calcium retention.[2]

Summarizing, it may be stated that rickets is a nutritional disturbance especially affecting the osseous, muscular, and nervous systems, with resulting lesions due to improper utilization of calcium and phosphorus.

Pathological Anatomy. In rickets the most characteristic changes are in the skeleton. The changes are distributed over the entire osseous system and become essentially manifest in those areas of the most active bone growth.

The following must be emphasized as characteristic factors of the rachitic bony process: hyperemia of the bones; irregular formation and proliferation of the tissues in which normally osseous formation occurs; deficient deposit of calcium in these tissues, and the pathologic decalcification of bones that already contain calcium.

The normal production of bone occurs in the periosteum and at the osteochondral borders, and here the essential anatomical factors of rickets are established.

The periosteum is for the most part decidedly thickened. The thickening is due to a hyperemic layer between the fibrous periosteal cover and the bone, consisting of alternate porous, plexus-like tissue and coarse longitudinal bands. Microscopic investigation reveals a substratum, rich in cells, which is interrupted by medullary vascular spaces.

Section of a normally growing bone reveals between the osseous and cartilaginous structures a limited and sharply-defined bluish zone of calcification several millimeters in thickness. Histologically the bone formation appears about as follows: Cutting centerward from the

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[1] Hamilton, B.: Am. Jour. Dis. of Children, 20—316, Oct., 1920.
[2] Tada: Monatschr. f. Kinderh., 1905—06, iv, 118.

cartilaginous epiphysis we find that the cartilage cells, which were at first deposited irregularly, arrange themselves in definite rows corresponding to the direction of growth, and begin to enlarge. The intermediary substance of these cell columns takes up lime salts and then becomes the zone of provisional calcification. From points of vascular ossification in these layers of cartilaginous proliferation capillaries originate which cause resorption of the cartilaginous tissue and form medullary spaces, in the walls of which, by proliferation of cells introduced through the vessels (osteoblasts), true bony tissue forms and soon becomes a compact structure by deposition of calcium salts.

In well-developed rachitic bones these conditions are essentially changed. The osteochondral boundary is markedly widened—to 1 cm. in thickness—and conspicuously red. In contrast to the normal cartilage the end is not sharp but serrated. As in the normal bone, the cartilaginous cells are first arranged in rows and afterwards clump together, forming larger nests. The enlargement of the cartilage cells, however, is much greater. The lines of direction are less conspicuous the closer we approach to the diaphysis. The proliferating capillary branches permeate this "chondroid" tissue much more deeply than in the normal bone, so that in place of a uniform approximation of the cartilage columns and medullary spaces there is a quite irregular interlacing of these histologic elements. Of essential importance in rickets is the fact that through the action of osteoblasts a structure arises which resembles bony tissue; this osteoid tissue, however, as was seen in the periosteum, differs micro-chemically from true decalcified bony tissue and shows but slight tendency to calcification.

Therefore, in the enchondral ossification, there is an enlargement of the zone of transformation, a marked hyperemia of the same, as well as the formation of osteoid tissue and delayed calcification. The last factor is

of essential importance in rickets. There is a conspicuous disproportion between the broad transitional zone, which is ready for ossification, and the slight deposit of calcium which is found there. The consequences of this is an abnormal softness at the epiphysis and a ready separation of the epiphysis from the diaphysis.

The bones are markedly hyperemic, alike in the periosteum, in the zones of ossification, and in the marrow. Through the great proliferation and the deficient calcification of the periosteum, as well as from the absorption of bone, they are abnormally soft. This gives rise to the many deformities and curvatures of the skeleton with which we have become familiar from the symptomatology. In addition there may be infractions and fractures which affect the shape of the bones of the extremities and the clavicle. A further consequence which has been mentioned is the softness and protuberance of the osteochondral borders of the long bones. Finally, there is an irregular periosteal deposit and by softening processes in the flat bones of the skull an irregular thickening and thinning is produced, the borders of the bone being thickened, the thinness affecting principally the squama of the occipital bone and the dependent portions of the parietal bones, in which probably unfavorable conditions are brought about by opposing pressure of the brain and the overlying surface.

In this series of bony changes all of those anomalies of the skeleton may be included which are met with in rickets: Craniotabes, the rosary, narrowed pelvis, and finally, the curvatures and deformities of the thorax and extremities.

The changes which the skeleton shows after healing of the process are better understood. After the zone of proliferation in the osteoid tissue is finally impregnated with lime. salts and has undergone ossification, the bone becomes thicker, harder, distorted, and its surface beset with rough edges and osteophytes. This condition is

especially distinct in the tubular bones, which lose their graceful contour, become heavier, and sometimes retain the shape which was produced in the florid stage by curvatures and infractions. These thickenings also occur in the cranial bones. The influence upon the permanent teeth, the pelvis and the longitudinal growth of the tubular bones has already been mentioned.

A very interesting question, although at this time obscure, is whether the organs of the body, as well as the skeleton, are affected in a characteristic manner by the rachitic process. This pertains especially to the brain,

Fig. 12.—D. V. First stage of rickets. Taken March 28, 1921, before beginning of phosphorized codliver oil therapy. The metaphysis is pale and hazy. The epiphysis of the upper end of the shaft of the tibia is not visible and at the lower end of the shaft is seen as an indefinite shadow. The periosteum appears as if separated from the shaft by a layer of osteoid tissue.

the spleen, the blood, the liver, and perhaps the muscles; the frequent pathologico-anatomical findings in the lungs and in the heart are of a secondary nature, and in the digestive tract no regular form of affection is noted. It is true the anatomical findings in the organs mentioned are not well-defined, and are by no means characteristic of rickets, but the relatively common occurrence of megacephalus and enlargement of the spleen and of the

liver, as well as the rarer hypertrophy of the brain, in connection with the rachitic fundamental process cannot at once be ignored.

Schmorl,[1] in his studies of healing rickets, found that the initial deposition of calcium occurring at the cartilage-shaft junction of the long bones takes place not throughout the rachitic metaphysis or at random in it but on the epiphyseal side of the metaphysis in that zone of the proliferative cartilage in which calcium deposition normally occurs and presumably would have occurred had rickets never been present. Figs. 12, 13, 14.
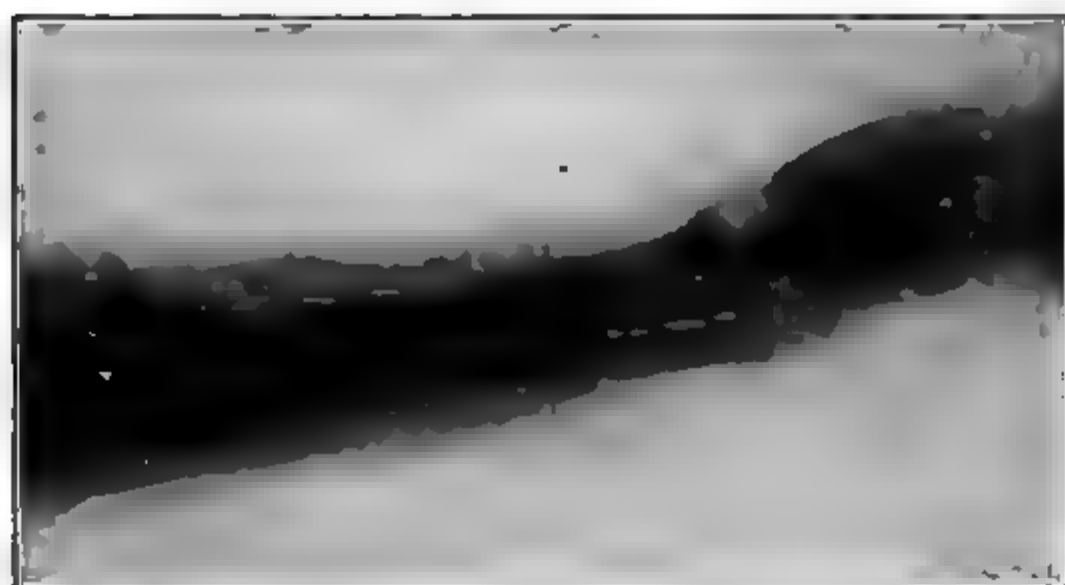
Fig. 13.—D. V. Taken April 26, 1921 after 28 days treatment with phosphorized codliver oil and a well balanced diet. A line of deposition of calcium salts is seen in the proliferating zone of the cartilage, at right angles to the long axis of the shaft. This deposit is most marked on the epiphyseal side of the metaphysis. The epiphyseal nucleus at the upper end is plainly visible and the one at the lower end shows an increased shadow density.

Recent investigations by Shipley, Park, McCollum, Simmonds and Parsons,[2] on the results following the feeding of codliver oil to rachitic rats, confirmed these

[1] Schmorl, G.: Ergebn. d. inn. Med. u. Kinderh., 13— 403, 1914.
[2] Shipley, Park, McCollum, Simmonds, Parsons—Jour. Biol. Chem., xlv, 343, 1921.

findings. They found that when the cartilage had become free from calcium as the result of the deficient diets, the addition of codliver oil to the food for a period of from two to seven days was followed by deposition of lime salts between the cells of the proliferative zone of cartilage. The deposit of calcium salts is linear, the width of the line apparently depending on the length of time during which the animal has been fed codliver oil. The line of deposition is at right angles to the long axis
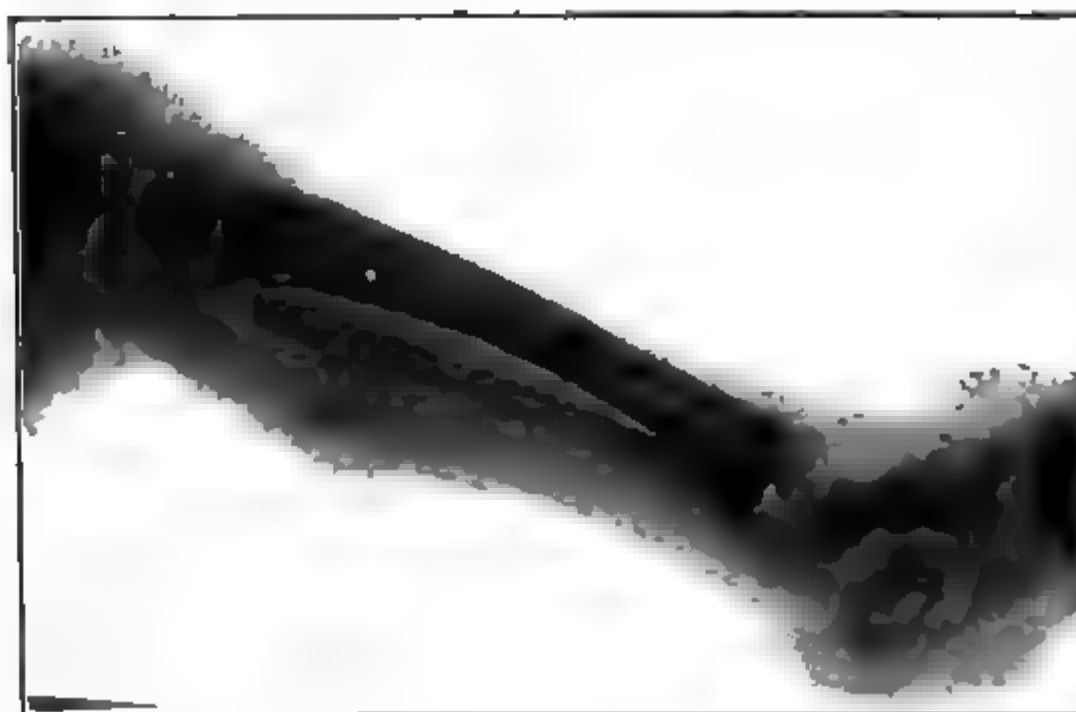
Fig. 14.—D. V. Taken May 11, 1921, 43 days after Fig. 14. Shows an increased length of the shaft due to calcium deposits at the epiphyseal side of the metaphysis. The increased length of the shaft at the lower end is about 2 mm. The nuclei at the upper and lower ends of the shaft are now distinctly visible and clean cut. Calcium has been deposited in the subperiosteal osteoid tissue and there is no longer the appearance of periosteal separation.

of the shaft of the bone. From the appearance of their sections it seems that very little lime salt is laid down in the osteoid tissue until calcification of the proliferative cartilage is complete.

The gross pathology in *premature* infants differs somewhat from that in mature infants. Megacephalus is

commonly observed. Along with this condition there is a symmetry of the skull which is produced mechanically by pressure on the infant's especially soft skull in the first months of life. The rachitic rosary is very prominent in prematures and is explained on the basis of the constant respiratory movements leading to deformities and marked enlargement of the epiphyses of the ribs. The chest is narrow and early shows the rachitic grooves. The long bones, however, only show moderate enlargements of the epiphyses in prematures, although rachitic changes appear very early in these bones. The bone absorption and fringing of the epiphyseal line with marked haziness at the end of the shaft, predominate rather than the increased proliferation which is the rule in full term rachitic infants. Probably the explanation for this is that the rickets appearing very early is terminated before the infant crawls or walks and so the compensatory proliferations from pressure and weight-bearing are not present. Consequently rickets may occur in prematures without the epiphyseal enlargement and curvatures of the long bones. The histological examination shows the characteristic picture, however, even in the absence of marked external manifestations.

Symptoms. The outstanding symptoms of well developed rickets will not be described here. It is, however, very important to be thoroughly familiar with the early symptoms of rickets, as in the first stage, before permanent damage has been done, it responds most readily to treatment. The first evidences of rickets may escape attention unless the examiner considers the possibility of its presence from the history. Fretfulness, irritability, restless sleep and excessive perspiration about the head are among the earliest signs. These children are usually pallid and show considerable evidence of a secondary anemia. Constipation frequently appears early and may alternate with diarrhea. There is soon noted

a more or less evident backwardness in physical development; the child may be unable to hold up its head and, at later age, to sit up or stand as a normal child would at the same age. (Fig. 15.) The muscles are flabby, the abdomen distended and tympanic, and an umbilical hernia is a frequent complication. (Fig. 16.) As the condition advances the anemia becomes more marked

Fig. 15.—H. G. Colored infant, showing an extreme degree of rickets. The head, extremities, chest and abdomen show the typical deformities. A large umbilical hernia is present.

and there is a tendency towards splenic enlargement. Among the earliest changes in the skeleton is the development of beading of the ribs at the costochondral junctions forming the socalled rachitic rosary. In the severer types this is followed by a sinking in of the ribs in the axillary line and a flaring out of the ribs below, the latter being due to the support furnished by the

Fig. 16.—H G   Same infant, showing the descending colon, sigmoid and rectum distended by a barium enema.   The megalocolon and enormous sigmoid account to a large extent for the abdominal enlargement and constipation in this case.

intra-abdominal organs. The depression at the costo-
chondral junctions, described as Harrison's Groove, is
to a large extent due to the traction on the chest wall
by the diaphragm. The skull usually shows enlarged
veins, and is squared in front, flattened on top, and has
marked frontal and parietal eminences. The fontanels
are late in closing, as are the sutures. Craniotabes is
one of the pathognomonic findings. At the junction of
the epiphyses and diaphyses nodular enlargements be-
come palpable in the long bones and are most easily
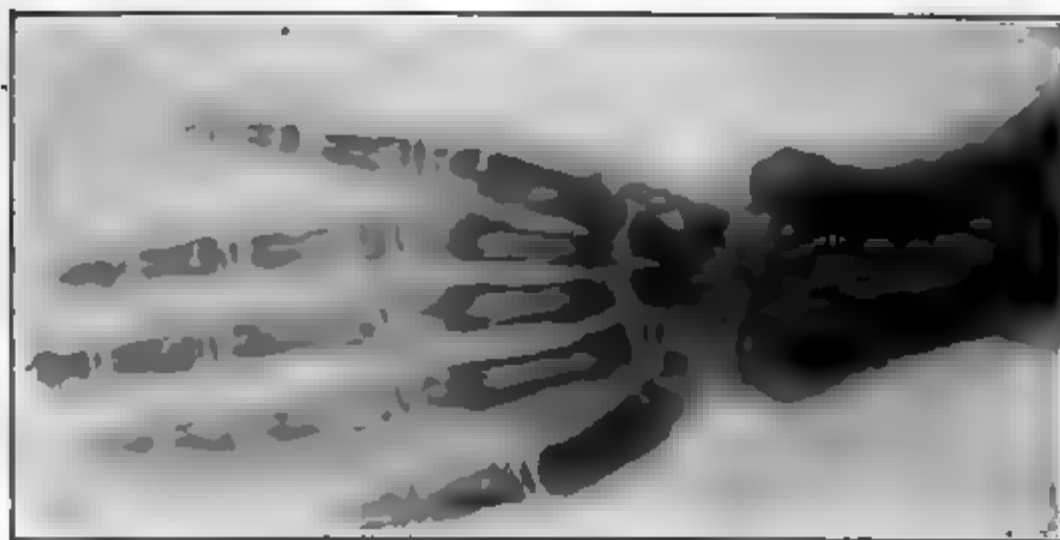
Fig. 17.—H. G. Same infant, showing the extreme changes
of a florid rickets. The typical enlargement of the soft parts is
noticeable in the outline of the wrists. The lower ends of
the shafts of the radius and ulna show the typical cupping
and sawtoothed appearance. The epiphyseal nuclei are widely
separated from the shafts and the nucleus of the radius is
displaced and fragmented. Similar changes are seen in all
of the metacarpal bones and the phalanges.

recognized at the wrists, knees, and ankles. (Fig. 17.)
Dentition is frequently delayed and irregular. The
primary teeth are subject to early decay. Deformities
are common and may develop in any part of the osseous
system, most commonly involving the long bones, verte-
bral column, and pelvis, as well as the head, previously
described. The blood changes are not characteristic,
usually being those of a secondary anemia, with a marked

reduction in the hemoglobin and red blood corpuscles, and not infrequently a moderate leucocytosis is present. The nervous phenomena, secondary to rickets, are among the most important from a therapeutic standpoint because of their reaction to proper treatment. They include all of the findings described under spasmophilia.

Of the greatest importance are the radiographic studies of the first and second early stages. These have already been described in the discussion of the gross bony pathology. These changes allow of a diagnosis often four to six weeks before the disease results in sufficient bone changes to be evident on physical examination.

Radiographic Diagnosis. Radiographic studies closely follow the macroscopic appearance of the bones. In the *first stage* the epiphyses cast little or no shadow, while the center of ossification is small or absent and at times appears multiple. These epiphyseal findings are easily explained ·by the interspersing of cartilage masses between the newly deposited osseous tissues, and are visible upon microscopic examination of fresh bone sections and often on macroscopic examination. The diaphysis becomes frayed out, instead of clear-cut, the periosteum thickened, and the joints appear hazy.

In this stage a wide separation of the epiphysis from the diaphysis is frequently noted, due to the lack of bone deposit in the proliferating zone or metaphysis. Multiple fractures are common.

In the *second stage* the shadow of the epiphysis becomes more marked, and the epiphyseal line is more ragged and irregular. The metaphysis is widened with an irregular saw-toothed appearance on the epiphyseal side and there is also a broadening and flaring of the epiphyseal line which is very characteristic. This continues into the third stage, and the space between the proliferating zone and the epiphyseal nucleus is lessened by a distance corresponding to the new bone deposit and epiphyseal growth. This growth can be measured on the

plates. Further changes in the second stage consist in the chambering of the interior of the bone, where light areas in the shaft indicate the absence of bone deposit, and heavier lines of ossification show the irregular development of trabeculæ. There is usually a thickening on the concave side of the shaft, which is a compensatory change. The second stage is generally a period of systemic reaction to the disease, in which signs of returning ossification occur and when deformity begins. In the *third stage,* the epiphysis begins to resume its normal contour and homogeneous shadow density. Irregularities persist in the marginal outline, and there is still a little mottling in the ossification. The lipping of the diaphyses has enlarged the bone ends, and there is in consequence a discrepancy in breadth between the diameters of the diaphysis near the epiphyseal line and the epiphysis.

These bony changes and the softening of the bone gives rise to all the various bony deformities of clinical rickets.

Prognosis. The disease is chronic, and lasts for months or up to the end of the first dentition. Its course is modified by treatment. Periods of latency and exacerbation depend on diet and hygiene and are influenced by season and secondary infections. Usually, active symptoms subside when a mixed diet is given—that is, at about the end of the first year.

Different parts of the skeletal system are progressively involved, recovery taking place in one while another is becoming affected. In the first six months of life craniotabes, beading and enlarged epiphyses may be the only physical signs. Later on the thorax becomes deformed, and then kyphosis and curvatures of the bones develop. The signs of improvement are diminished sweating and restlessness, disappearance of craniotabes and anemia, increasing muscular power, and improved general nutrition. Deformities slowly and steadily im-

prove except in very bad cases, in which they persist to a variable degree throughout life, in the shape of pigeon-breast, Harrison's groove, eversions of the costal arch, kyphosis, pelvic deformity, knock-knee, bow-legs, and flat-foot.

Rickets is never fatal *per se,* but it reduces the resisting power, and is an important cause of increased mortality from other diseases, especially catarrhal affections of the alimentary and respiratory systems. Death may also result from spasmophilic manifestations, more especially convulsions. The thoracic deformity retards growth by interfering with efficient lung expansion and oxidation. Impaired epiphyseal growth may affect growth in height, and the malnutrition may delay mental development. Rachitic children often talk late and learn new words slowly.

Treatment. In a consideration of the treatment of rickets prime importance must be placed upon a careful study of the previous diet and life history of the infant. *First,* in a consideration of the preceding diets, the cases may be classified into those which have been wholly or largely *breast-fed.* In this group the general health of the mother and her diet must be investigated. The quantity of her milk should be estimated. Unfortunately the quality can usually only be judged by the effect on her offspring. The *second* group of cases are those dependent upon *cow's milk as the main article in their diet.* In this group the source and quality of the milk as well as the quantity must be considered. The diet usually errs in the direction of insufficiency, however, an excess of cow's milk may result in a disturbed metabolic balance with a secondary disturbance in calcium retention. The *third* group of cases are those dependent upon *proprietary infant foods which contain cow's milk.* The danger in feeding those containing cow's milk usually lies in the fact that the quantity of cow's milk they contain is insufficient, or its chemical composition is

changed in its preparation, and its vitamine content destroyed by heating and ageing. The caloric value in this class of foods is largely dependent upon the carbohydrates contained and added while their fat and protein is usually low. The *fourth* group are those fed on *proprietary foods containing little or no cow's milk* and which are composed largely of dextrinized cereals which have been devitalized through long continued heating and are dangerous because of a second factor, namely, the instruction to feed them with an insufficient quantity of cow's milk and other needed food elements.

Breast feeding must be encouraged. The lactating mother's diet should be rich in milk, eggs, butter and green vegetables. These may be supplemented by cereals, root vegetables and muscle-meat, but it is to be emphasized that the latter group are decidedly inferior to the former in the furnishing of needed minerals (especially calcium and phosphorus) and vitamines in the breast milk.

If the breast milk becomes deficient, either qualitatively or quantitatively, mixed feeding must be instituted.

In *artificially fed* infants the disease is usually present during the second quarter of the first year, although it may not be clinically manifest until a much later period. A proper diet at any age should contain sufficient fat, protein, carbohydrate, salts and vitamines. In most instances, cow's milk must in greater part furnish these necessary elements. The cow's milk should be supplemented as soon as possible by the addition of fruit juices and codliver oil. Fresh cereals, in the form of well-cooked gruels, can usually be added by the fourth month of life and fresh vegetables in the form of a vegetable-cereal-meat soup, can be started in the sixth month. Vegetable purées may be added by the eighth or ninth month. The vegetables, whether contained in soup or when given as purées, should be passed through a fine sieve. The addition of fruit juices, cereals and vege-

tables should be considered in the light of prophylactic treatment against rickets. The careful study of the whole diet should be undertaken and a generous diet, as above outlined, should be instituted as routine in the feeding of all infants.

Not infrequently the diets of cases developing rickets are also low in the anti-scorbutic element, making the early addition of fruit juices and cooked fruits of additional value. In the formulating of the diet in these cases, the presence of the secondary anemia which so commonly accompanies rickets must be considered and the iron containing foods should be added in sufficient quantities. For this purpose the green-leafed vegetables and meat juices or scraped meat pulp are of great value. Therefore, in rickets a varied diet may be instituted in every case as soon as it can be utilized. However, the new food elements must be added one at a time and subsequent changes made only after it has been ascertained that the infant can digest the previous diet. Sudden and radical changes in the diet may lead to serious gastro-intestinal and systemic complications.

Hygienic Treatment. The infant should receive plenty of fresh air and sunshine. The practical value of the direct action of the sun's rays in the treatment of rickets has been conclusively demonstrated. The weather permitting, they should be outdoors for several hours a day, varying the period according to the weather.

"Mothering" in the form of exercise, handling and light massage, are invaluable to all infants. This is especially true of infants in institutions.

The susceptibility of rachitic infants to respiratory infections must be remembered and every means employed to prevent systemic depression due to exposure to rapid changes of temperature. Because of the increased susceptibility of rachitic children to infections, they should be protected as far as possible from any contact

with infectious cases. Slight colds may result in serious systemic infections.

Sunlight. Experimental evidence showing the favorable action of sunlight on the mineral metabolism was furnished by Raczynski[1] in 1912. He showed that in rickets a deposition of salt in the bones may be accomplished without any addition or alteration in the diet. He took two puppies of the same litter, both of which were being suckled by the mother, and kept one in absolute darkness and the other, throughout the day, in sunlight. At the end of a six weeks' period both were killed. An analysis of their bodies showed that the one which had been reared in the sunlight contained over 50 per cent. more calcium and 25 per cent. more phosphorus than the other, but that on the contrary, it contained less than half the quantity of chlorin.

Powers and his co-workers[2] in a series of experiments on rats, using a diet which in ordinary room-light gave rise to a disease in its essential features identical with rickets as seen in human beings, used a diet high in calcium, low in phosphorus and insufficiently supplied with fat-soluble A. In other respects it was well constituted. All control rats kept in room-light developed rickets, while rats exposed regularly to sunlight on an average of four hours a day remained, without exception, entirely free from rickets. The animals, however, remained under-sized; the bones, though completely calcified, remained thin. These experiments lead them to believe that even in the presence of a defective diet, the sunlight raises the efficiency of the body cells. The favorable effect of sunlight in rickets has recently been emphasized by Feer.[3] He calls attention to the marked benefit which accrues in rickets from exposure to the

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[1] Raczynski, J.: Compt-Rend. de L'Ass'n., Internat. de Pediat., Paris, 1913, 108.

[2] Powers, Park, Shipley, McCollum and Simmonds: J. A. M. A., 78, 3, January, 1922.

[3] Feer, E.: Schweiz. med. Wchnschr., 51, 438, 1921.

sun's rays in the Swiss Alps. Riedel[1] reports a series of cases treated with sunlight on bright days and supplementing the treatment with a quartz lamp on sunless days, with excellent results.

Huldschinsky made use of sunlight together with the ultra-violet rays in some of his series, and Riedel relied on treatment with sunlight. Hess and Unger[2] demonstrated, by means of the roentgen ray, that sunlight possesses a curative action in rickets of human beings. They exposed five infants with rickets to the direct action of sunlight for periods varying from one-half hour to several hours daily, whenever the sunlight was available. Different parts of the body were in turn subjected to the action of the sun's rays. In one of the cases the patient was exposed to the sunlight only on seven occasions, for a total period of twenty-five hours. The general condition of the infant's health, as well as the diseased condition in the bones, was benefitted.

Ultra-violet Rays. Since Bucholz,[3] in 1904, reported his good results in the treatment of rickets by artificial light and heat, by exposure to the rays of a special lamp, little attention has been paid to this form of therapy until the publication of the work of Huldschinsky,[4] in 1919. The latter made use of the quartz lamp. In December, 1918, Winkler[5] reported the favorable effects of treatment of rickets with the roentgen ray. He used a medium soft tube at a focal distance of about 20 cm. The exposure did not exceed ninety seconds and was repeated every other day. The treatment at first was directed against the craniotabetic lesions of the head.

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[1] Riedel, G.: Munchen med. Wchnschr., 67, 838, July, 1920.

[2] Hess, A. F. and Unger, L. J.: Amer. Jour. Dis. of Children, Aug. 1921, 186.

[3] Bucholz, E.: Verhandlung der Gessellschaft für Kinderheilkunde in der Abteilung fur Kinderheilkunde der 76 Versammlung der Gessellschaft Deutscher Naturforscher und Aerzte in Breslau, 21, 116, 1904.

[4] Huldschinsky, K.: Deutsch. med. Wchnschr., 45, 712, 1919.

[5] Winkler, F.: Monatschr. f. Kinderh., 15, 520, Dec., 1918.

After five or six treatments, he observed that the sweating of the head came to an end and sleep was improved. As the treatment progressed, laryngospasm and the tendency to convulsions disappeared. The craniotabes vanished, the teeth erupted and the calcium deposition occurred at the end of the long bones. In 1920 Putzig[1] used the quartz lamp in the treatment of rickets in premature infants, with good results. In 1920 Riedel[2] and in 1921 Erlacher[3] reported further cures with the use of the ultra-violet rays, and in 1921 Mengert[4] announced the successful use of the quartz lamp as a prophylactic agent against rickets. He claims it to be especially good in the prophylactic treatment of prematures. In 1921, Hess and Unger[5] also reported the cure of rickets by means of the ultra-violet rays. They report a group of infants, varying in age from 8 to 21 months, which were treated during the months of February and March, the period of the year when rickets is likely to manifest its highest incidence and least apt to decrease in severity. During the period of treatment no changes were made in the diet. Marked improvement in the bones was demonstrated by means of roentgenograms, and the hemoglobin percentage and number of red cells increased in every instance. The infants were treated three times a week for a period of two months. The entire body was exposed to the rays, at first for a period of about three minutes, the length of exposure being increased gradually to twenty minutes.

Kramer, Casparis and Howland[6] found that by systematic exposure to the rays from the mercury vapor quartz lamp the inorganic phosphorus concentration of the

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[1] Putzig, H.: Therap. Halbmonatschr., 8, 234, April, 1920.

[2] Riedel, G.: Munchen med. Wchnschr., 67, 838, July, 1920.

[3] Erlacher, P.: Wien. klin. Wchnschr., 34, 241, May, 1921.

[4] Mengert, E.: Deutsch. med. Wchnschr., 47, 657, June, 1921.

[5] Hess, A. F. and Unger, L. J.: *Ibid*, 13.

[6] Kramer, B., Casparis, H., Howland, J.: Amer. J. Dis. of Children, xxiv, 20, 1922.

serum of these children which was low (from 2.7 to 3.2 mg.) before the treatment was begun gradually increased to a maximum of 6 mg. with the appearance of calcium deposition in the bones. So far as could be judged, healing of the bones following radiation occurred at about the same time as it does after the administration of codliver oil. The changes in the phosphorus concentration of the serum were identical with those observed after codliver-oil treatment.

It is my practice to begin with 50 cm. distance for three minutes, gradually increasing the time of exposure to twenty minutes by the end of the second month. At least three treatments should be given each week—when possible they should be given daily. The entire body should be exposed, the ventral and dorsal aspects being radiated during alternate treatments. The eyes must be protected by suitable glasses.

In using the quartz lamp in practice I apply the following exposures as suggested by Gerstenberger:

1st Month:
TOTAL EXPOSURES 12.
At 80 cm. for 5, 7, 9, 11 minutes.
At 75 cm. for 7, 9, 11, 13 minutes.
At 70 cm. for 9, 11, 13, 15 minutes.

2nd Month:
TOTAL EXPOSURES 12.
At 70 cm. for 15, 17, 19, 20 minutes.
At 65 cm. for 15, 17, 19, 20 minutes.
At 60 cm. for 15, 17, 19, 20 minutes.

The time and distance requirements should be observed in detail in order to avoid an unnecessary dermatitis and also to make possible a rapid increase in exposures.

If no inconvenience or discomfort is caused to patient divide the time of exposure into halves—one for the anterior surface of the body and one for the posterior surface.

Any developing dermatitis must be treated by application of bland lanolin ointment.

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In summarizing the *curative* value of light—sunlight and artificial—we may say with positiveness when either one of these or both are made available to a rachitic infant, the defense mechanism which has previously been ineffectual is put in operation. Healing is frequently seen, even in the absence of a change in diet, and therefore, in cases so treated cannot be due to the supplying of the body with either calcium or phosphorus but must be dependent upon the raising of the potential of cellular activity which results in a more efficient utilization of the salts which are directly or indirectly concerned with ossification and calcification. Both methods of treatment result in an increase in the calcium and phosphorus content, the hemoglobin and the number of red cells in the blood.

The quartz lamp can have only a limited application as a therapeutic agent in general practice. The greatest value of the experimental researches with ultra-violet rays lies in the affirmation of the earlier good reports with heliotherapy and the relation of certain light rays to the normal metabolic processes of the human organism.

If no other service is rendered they should at least lead to the more general recognition of the necessity of sunlight in the promotion of body growth and development. This means that good hygiene is essential to health and is of especial importance as a prophylactic measure against rickets in young infants, and an absolute essential to its cure.

Medicinal. Although there is still much conjecture regarding the fundamental etiology of rickets, yet some pretty definite facts are known regarding the medicinal therapy.

It has been repeatedly demonstrated that codliver oil will prevent rickets and increases the amount of calcium and phosphorus retention in rickets, although this action may be slow in the florid stage. As mentioned previously, Howland and Park have very recently demon-

strated anatomically in animals a beginning calcium de-posit in the bones two days after beginning the administration of codliver oil, and in three weeks they were able to demonstrate similar changes in infants by means of the roentgen rays. Combining phosphorus with codliver oil makes it more efficacious. As previously stated, Phemister, Wegner and Kassowitz have shown that phosphorus, when administered without codliver oil, stimulates bone growth and increases the calcium deposits in the healthy bones of normal growing children and also in those rachitic. The efficacy of administering calcium to rachitic patients is open to much question. Most investigators believe it is of no value except possibly in the case of prematures. Sufficient calcium is taken with the food, the trouble being rather one of poor calcium retention. During convalescence, when the retention of calcium is extreme, calcium administration may be of assistance. One of the best preparations is a 10 per cent. tricalcium phosphate C. P. in an emulsion of cod-liver oil U. S. P., which makes a smooth suspension and is readily taken by infants.

To be most effective codliver oil, with or without phosphorus, should be started early in the artificially fed and also the breast-fed of races or individuals showing a tendency to development of rickets in the offspring. This is especially true of the Italian, Jewish and Negro races. Prematures, even when fed upon breast milk, show a marked tendency toward development of rickets, and should be placed upon codliver oil and phosphorus treatments not later than the second month. It is our custom to start phosphorized codliver oil, gr. $\frac{1}{200}$ of phosphorus to one dram of codliver oil (0.0003 Gm. to each 4 mils), in small doses, by the end of the sixth week in infants artificially fed on heated milk mixtures. At first 0.3 to 1.0 mil (5-15 minims) is given once or twice daily. The dose is rapidly increased until by the end of the third or fourth month, 2 to 4 mils ($\frac{1}{2}$ to 1 dram) are

given twice daily. Following the sixth month, 4 mils (1 dram) may be given two or three times daily. We find in most cases it is well taken when given in orange ·juice, the anti-rachitic and anti-scorbutic combination being an excellent one in the artificially fed. The codliver oil therapy should be continued well into the time of feeding of a general diet containing more especially animal fats, cereals and vegetables. The anemia must be combated with iron preparations. (See Secondary Anemia, page 414.)

Deformities. Development of deformities of the osseous system, more especially of the spine and long bones, can to a large extent be prevented. Infants should be discouraged from walking until the bones are firm. Head deformities can be lessened by changing the position in which the infant sleeps. Scoliosis may be avoided by preventing the child from bearing weight on the spine through early attempts at sitting, and improper holding and carrying of the infant, bowing and distortion of the legs by preventing crossing of the legs around the potbelly. Coxa vara is due to the weight of the trunk. Many of the deformities can be avoided by the institution of the proper orthopedic measures. Massage is of great value in strengthening the weakened muscles.

PART VI.

Spasmophilia.

(Spasmophilic Diathesis. Infantile Tetany.)

THE term spasmophilic diathesis, as applied in the clinical sense, refers to a constitutional anomaly characterized by a general hyperexcitability and irritability of the nervous system. The most frequent *active* manifestations are general convulsions, laryngospasms, spasmodic apnea and carpopedal spasm. Among the *latent* manifestations are Chvostek's facial phenomenon and Trousseau's sign. Among the most constant findings is Erb's sign, the presence of hyperexcitability of the peripheral nerves, evidenced by reaction to the galvanic current.

Etiology. *Heredity and familial predisposition* have in the past been considered as important factors. In the light of our present knowledge as to changes in the body chemistry in this condition, their relationship is open to question; however, it is our belief that certain types of infants show symptoms of an unstable nervous system even from birth and are therefore more likely to develop active clinical manifestations. To this class of cases belong the restless, nervous infants with a tendency to pylorospasms and repeated vomiting. The colicky infant which is often overfed to pacify it, thereby resulting in the development of nutritional disturbances, belongs to this group. They are often hypersensitive to light and sound, often precocious, and require much entertaining and soon become little tyrants, seemingly ruling the household. They form bad habits readily and the utmost care must be taken to train them properly as to

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regular habits of eating and sleeping. Added to the difficulties of these infants is the presence of neurotic parents, who often show little inclination to train them. We must, however, not make the error of believing that most infants developing spasmophilia during their infancy have this hereditary tendency.

Spasmophilia has in the past been described as a clinical entity. In the light of more recent experimental and clinical facts, it must be regarded as a *symptom-complex* without a specific etiology. Tetany may be produced experimentally by the excision of the parathyroid glands, by the injection into the blood stream of sodium phosphate or of sodium bicarbonate, by diets high in potassium and sodium phosphate, and by depleting the system of necessary salts. It occurs spontaneously in many cases of rickets. It may be likened to uremia in the course of nephritis.

Rickets. As a result of their experiments, Shipley, Park, McCollum and Simmonds[1] were led to believe that there are two main kinds of rickets. One is characterized by a normal or nearly normal blood calcium and a low blood phosphorus (low phosphorus rickets); the other by a normal or nearly normal blood phosphorus but a low blood calcium (low calcium rickets).

If the hypothesis just stated is correct, they believe the relation of tetany to rickets would appear to be as follows: Tetany is essentially an expression on the part of the nervous tissues of an insufficiency of the calcium ion; rickets is essentially an expression on the part of the skeleton of disturbed relations between the calcium and phosphate ions of the body fluids. Tetany is frequently associated with rickets because rickets is a disease in which the calcium ion in the body tissues and fluids is subject to variations. Tetany may occur independent of manifest rickets. Since tetany may occur with the low

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1 Shipley, Park, McCollum and Simmonds: Am. Jour. Dis. of Children, v, 23, p. 91, 1922.

phosphorus form of rickets, it does not serve to mark off one form of rickets from the other. Tetany, however, is essentially associated with the low calcium form of rickets and, for all practical purposes, the low calcium form of rickets is the rickets of tetany.

While occasionally cases are seen in which clinical evidence of rickets is not demonstrable, these cases are on the whole exceptional. On the contrary, many cases of rickets, even very severe types, exist in which tetany does not supervene.

Diet. The *feeding* history, with its associated nutritional disturbances, is of great importance in the interpretation of the clinical manifestations. It is but rarely seen in the breast-fed, and most frequently seen in the infants fed upon proprietary foods, more particularly those who have had repeated changes in their diet. Overfeeding with a diet composed almost exclusively of cow's milk frequently aggravates the condition and may, in latent cases, precipitate the active manifestations in all probability due to a disturbance of the salt metabolism by the whey. content of the cow's milk. One of the most striking phenomena in the treatment of these cases is the rapid disappearance of all manifest signs when the infants are placed upon breast milk, with the reappearance upon the addition of relative excesses of whey or large quantities of cow's milk.

Nutritional disturbances. and various errors of diet often predispose to attacks. These errors of diet may be overfeeding (quantitative) or improperly proportioned diets (qualitative), which sooner or later result in acute nutritional disturbances which precipitate the clinical manifestations.

Premature infants evidence a marked predisposition, but in the breast-fed receiving sufficient food, the manifest symptoms occur exceptionally. Convulsions, when present in the breast-fed, usually follow acute nutritional disturbances and infections. Not uncommonly the mere

changing to cow's milk precipitates convulsions. Even in premature infants fed on human milk the electrical irritability often is such that C.O.C. is less than 5 milli-amperes at the age of 6 to 10 weeks. This electric hyper-irritability is most frequently seen during the fourth month in prematures and gradually disappears during the fifth to sixth month.—Yllpö.[1]

The early development of rickets in the premature may in greater part account for the early development of spasmophilia in these infants.

Acute Infection. Some form of infection, although slight, is frequently the precipitating factor of the acute manifestations. Reinfection will aggravate the symptoms or they may precipitate fresh attacks in healed cases.

Vitamines. The work of Mysenburg[2] tends to dis-prove any relationship between the vitamines and spas-mophilia.

Age. The active manifestations are usually evidenced between the sixth month and second year in full-term infants, while in the premature they may be seen as early as the second to the fourth month.

Season. The most active manifestations, such as con-vulsions, laryngeal and carpopedal spasms are most com-monly seen during the winter and early spring months. The season of highest instance in most cases corresponds with that in which rickets becomes most manifest. Poor hygiene, lack of sunlight, secondary infections in the infant, and, in all probability, the diet of the mother, or that of the cows, are all contributing factors. The convulsions seen with summer diarrhea are more often toxic or infectious in origin.

Pathogenesis. Several theories have been advanced. All of these have as a basis some fundamental disturb-

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1 Yllpö: Zeitschr. f. Kinderh., xxiv, 1919, 1.

2 Mysenburg: Am. Jour. Dis. of Children, V. 20, p. 206, Sept., 1920.

ance in metabolism. The most generally accepted has been based on experimental and clinical evidence of decreased calcium retention. More recently considerable data has been produced seemingly pointing to increased retention of the alkali phosphates (K and Na), with a secondary diminution of Ca salts as precipitating factors.

Kramer and Howland found that in children suffering from rickets alone, the phosphorus of the blood serum is low, and the calcium not far removed from the normal; in children suffering from manifest tetany complicating rickets, on the other hand, the calcium is low but the phosphorus not far removed from normal.

Calcium Metabolism. A calcium deficiency in the tissues has been demonstrated by numerous investigators, more especially in the brain and blood. The earlier investigations on the blood, by Howland and Marriott,[1] have more recently been confirmed by Kramer, Tisdall and Howland.[2] They found the calcium of the blood serum to be low in tetany, averaging 5.6 milligrams per 100 cubic centimeters of serum, about half the normal. Their lowest estimation was 3.5 milligrams per 100 cubic centimeters of serum. The average normal amount of calcium per 100 cubic centimeters in their cases was between 10 and 11 milligrams. The latter group of workers found the concentration of the sodium, potassium and magnesium in the serum of the patients with tetany is essentially normal. On the other hand the concentration of the calcium is regularly lowered. Obviously the important factor in the increasing irritability of the neuromuscular mechanism in infantile tetany is the decrease in the calcium concentration. The stimulating effect of the sodium and potassium salts is unopposed by the inhibitory effect of calcium.

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[1] Howland and Marriott: Quarterly Jour. Med., xi, 1917, 18. 289.

[2] Kramer, Tisdall and Howland: Am. Jour. Dis. of Children, xxii, 431, 1921.

The relation of calcium to the symptoms of spasmo-
philia has been studied extensively, especially its influ-
ence on the electric excitability. Physiologists have
shown that certain mineral ions exert a specific effect on
muscle nerve irritability. Rosenstern[1] and Sedgwick[2]
reduced the electric irritability in spasmophilic infants
by administering large doses of calcium by mouth.
Loeb's[3] findings indicate that Na and K increase the
threshold for excitation, while Ca and Mg tend to de-
crease this. This muscle nerve irritability is the func-
tion of the quotient $\dfrac{Ca \text{ plus } Mg}{Na \text{ plus } K,}$ as designated by Reiss.[4]
Holt[5] has demonstrated that in the course of diarrheal
attacks there is a much greater loss of Na and K than Ca
and Mg in the stools. Diuresis and catharsis often cause
an improvement in the spasmophilic symptoms.

Phosphorus Metabolism. Howland and Kramer[6] have
determined the inorganic phosphate of the serum in a
large series of normal infants. They found the con-
centration to average 5.4 milligrams per hundred cubic
centimeters, with a minimum of 4 milligrams and a
maximum of 7.1 milligrams, the higher values being
usually found in the serum of exclusively breast-fed
children. A determination of the inorganic phosphorus
of the serum in infantile tetany revealed that in about
half the cases the phosphorus concentration was within
normal limits or slightly above normal. This is in marked
contrast with cases of uncomplicated rickets, *i.e.*, with-
out tetany, in which they found a much reduced inor-
ganic phosphate serum content. In no case did they

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[1] Rosenstern: Jahrb. f. Kinderh. lxxii, 1910, 154.

[2] Sedgwick, J. P.: St. Paul, Med. Jour., 1912, Vol. xiv, 497, 519.

[3] Loeb, J.: Oppenheimer's Handbuch der Biochemie.

[4] Reiss: Zeitschr. f. Kinderh., 1911, iii, 1.

[5] Holt, Courtney and Fales: Am. Jour. Dis. of Children, 1915, ix, 213.

[6] Howland, J. and Kramer, B.: Am. Jour. Dis. of Children, xxii, 105, 1921.

find a marked increase in the inorganic phosphorus concentration above the normal level. On the whole, the inorganic phosphorus of the serum showed a considerable variation. In a number of cases there was a relative increase when compared with the calcium concentration. The significance of this relatively high phosphate content in the presence of decreased calcium in cases of rickets complicated by tetany is as yet not clear. They state that an increase in the inorganic phosphorus of the serum alone, however, does not seem to be responsible for infantile tetany.

Binger[1] was able to produce tetany by the intravenous injection of orthophosphates. He found that not infrequently in conditions with a calcium deficiency alone tetany was absent. These authors state that the reduction of calcium alone is not sufficient to bring about the symptoms of tetany unless certain other conditions are satisfied. The question as to the nature of such associated findings must be answered by further study.

Jeppsson and Klercker,[2] in a series of experiments, found that by feeding of 0.20 Gms. of P_2O_5, in the form of K or Na diorthophosphate per kilogram body weight, to the normal infant, and 0.10 to spasmophilic infants, they were able to produce symptoms similar to those seen in active spasmophilia or to activate the manifestations in latent cases (0.20 gram of P_2O_5 represents in alkali diorthophosphates 0.27 Gms. of K_2O and 0.18 Gms. of Na_2O or 0.49 Gms. of K_2HPO_4 and 0.39 Gms. of Na_2HPO_4). When using the potassium salts at times these results were manifested in a few hours. With the sodium salts larger quantities were required and often the symptoms were not evident until after two or three days of treatment.

They believe that most spasmophilic children receive an excess of alkali phosphates in their diets, more espe-

[1] Binger: Jour. Pharmacol. and Exper. Therap. 10, 1917, 105.
[2] Jeppsson and Klercker: Zeitschr. f. Kinderh., 1921, Vol. 28, 71.

cially during the first and second year, with the exception of the first few months of life, and that the alkali phosphates play a rôle in the genesis of tetany.[1]

The parathyroid theory is to a large extent based upon the fact that the physiologic and chemical findings in spasmophilia, in infants, and parathyroid tetany, in animals, are nearly identical. The histological evidence is conflicting and for the most part negative. Accidental removal of the parathyroid gland in humans and experimental excision of these glands in animals, have both resulted in a tetany that resembles in its clinical manifestations the spasmophilia of infants. Following the animal experiments Howland and Marriott[2] have demonstrated a diminution in the calcium content of the blood. These findings have been verified by MacCallum and his co-workers,[3] who also found a decreased calcium content in the brain and an increased excretion. They believe that the parathyroid gland regulates calcium metabolism and that failure in its secretion results in a lessened retention. Greenwald,[4] in his experimental studies, found that the phosphorus excretion in the urine of his animals was greatly decreased, to as low as 8 per cent. of the normal, shortly after operation but after development of tetany it increased rapidly, occasionally to an amount in excess of the preoperative content. He also found an increase of the phosphorus content of the blood before the appearance of tetany. There was also a sodium and potassium retention. He believes that fol-

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[1] J. K. Calvin and M. P. Borowski: Amer. Jour. Dis. of Children, 23, 1922, instituted a series of investigations on our service at Cook County Hospital to corroborate the Jeppsson and Klercker findings. They were unable to duplicate these results by the use of the dosage of potassium or sodium diorthophosphates recommended by these investigations. The observations were made on rachitic infants varying from 6 to 18 months of age, some of whom had recently recovered from actively manifested spasmophilias.

[2] Howland and Marriott: Trans. Amer. Ped. Soc., 28, 200, 1916.
[3] MacCallum and Voegtlein: Jour. Exper. Med., xi, 118, 1909.
[4] Greenwald, J.: Jour. Biol. Chem., Vol. 14, 370, 1913.

lowing the extirpation of the parathyroid there is a decreased excretion through the kidneys and an abnormal retention in the tissues of the alkali phosphates, which is followed by a decreased retention and an increased excretion through the kidneys as soon as the spasms develop.

There is, however, great question as to the relationship of parathyroid dysfunction and tetany in the infant. Pathological studies lead us to believe that parathyroid lesions in infantile tetany are the great exception. Parathyroid lesions have been described in patients who have shown no evidences during life of the pathognomonic findings of tetany.

In summarizing the pathogenesis we may state that a diminution of the calcium salts in all probability is the most important factor in the development of this condition. However, the possibility of an absolute or relative excess of the phosphates, especially the sodium and potassium salts, playing an important rôle cannot be overlooked. Abnormal ratios in the body salts, as in rickets, play an important rôle. The relationship of disturbances in parathyroid functions to the diminution of calcium tissue content must be made the subject of further study before its importance can be fixed. Infections are the most frequent factor in precipitating manifest symptoms in the latent cases. The seasonal incidence emphasizes the importance of the relationship between active rickets, infections and spasmophilia.

Symptoms. The term *spasmophilic diathesis* is used in a clinical sense to cover a symptom complex, comprising a group of signs, any one or all of which are pathognomonic of the condition.

They consist of two groups, which are best described as *latent* and *active*. Cases presenting all of the signs to be described are the exception and in most instances the diagnosis will be made in the presence of one or more of them.

The *latent signs* are more apparent than the active ones, and in their most frequent sequence are:

Erb's sign, better designated as increased electric hyperexcitability; *Chvostek's facial phenomenon*, and *Trousseau's sign*. Of the *active signs* generalized convulsions are the most frequently seen, while laryngospasm, spastic apnea and carpopedal spasms are of less

Fig. 18.—Spasmophilia—Carpo-pedal spasm. This infant was of the neuropathic type from birth and also presents a marked case of exudative diathesis.

frequent occurrence. It, therefore, becomes obvious that many cases of spasmophilia would be overlooked were it not for the uncovering of the latent manifestations during the course of a routine physical examination. On the other hand, the interpretations of convulsions must be dependent to a large extent upon a careful study of the clinical history. Otherwise, cases of spas-

mophilia will be overlooked or a diagnosis of this condition made in cases due to other causative factors.

Erb's Sign (Increased Electrical Irritability). The test is made with a galvanic current, and for this purpose a small battery made up of dry cells answers the purpose best for the finer tests. However, there are batteries with transformer attachments which can be used with ordinary lighting current. A large, flat electrode (5 cms.) is placed on the chest or upper abdomen of the infant and a small Stintzing electrode, 1 or 2 cms. in diameter, is placed over the median nerve, just below the elbow, or over the peroneal nerve in the outer part of the popliteal space near the head of the fibula. For the purpose of making the test the opening contractions are used.[1] The C. O. C. is usually first used because of its being the easier of the two opening contractions to demonstrate, due to the fact that in normal infants under 2 years of age, approximately nine M. A. are required to obtain a reaction. After completing this test the pole should be reversed and the A. O. C. tested. In making these tests the C. C. C. is of little value and the A. C. C. is only used for comparison with the A. O. C. The test, whether studying the C. O. C. or A. O. C., should always be begun with a current of sufficient strength to produce a contraction of the muscles of the palm and fingers, when the median nerve is used, and the muscles of the dorsal surface of the foot and toes when the peroneal nerve is used. (Figs. 19 *a, b.*)

The average normal reaction, under 2 years of age, will approximate the following:

C. C. C.	A. C. C.	A. O. C.	C. O. C.
2	3	5	9

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[1] For purposes of convenience the following terms have been abbreviated:

Cathodal closing contraction .... C. C. C.
Anodal closing contraction .... A. C. C.
Anodal opening contraction .... A. O. C.
Cathodal opening contraction .... C. O. C.
Milliamperes .... M. A.

In infants the following electrical reactions are pathognomonic of tetany, in the absence of brain lesions.

C. O. C. under 5 M. A.

A. O. C. with less current than that causing an A. C. C. and under 5 M. A. up to the end of the second year. After this age there is a normal tendency for the A. O. C. to appear with less than 5 M. A.

A C. O. C. with less than 2 M. A. is indicative of the
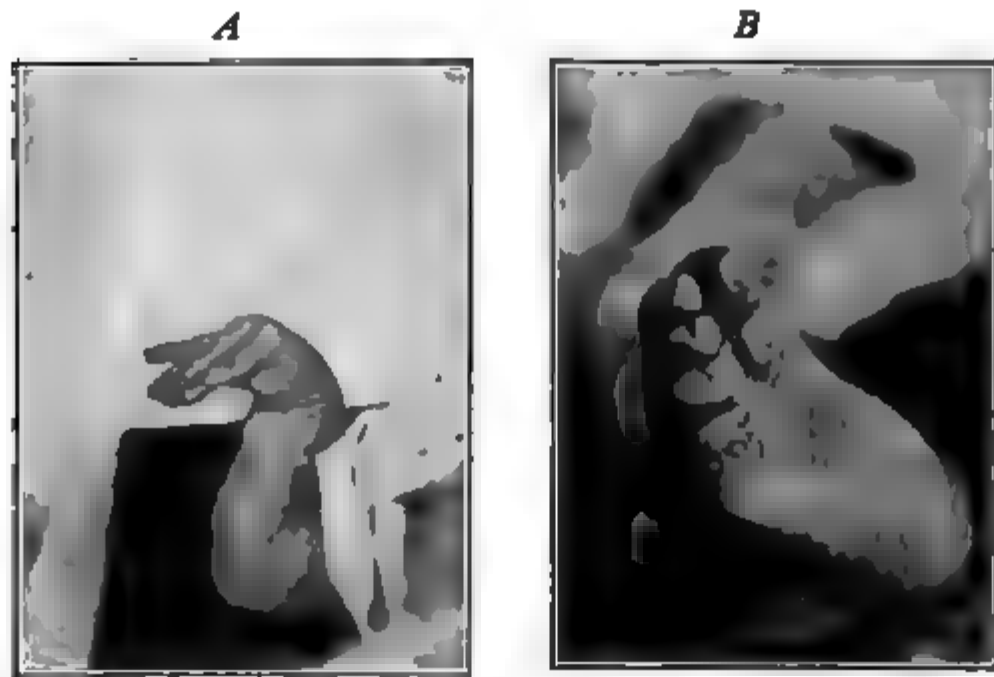
Fig. 19.—Spasmophilia. *A.* Carpal Spasm. *B.* Pedal Spasm.

appearance of early convulsions and should always be considered as indication for active treatment.

Chvostek's facial sign depends upon the hyperexcitability of the facial nerve. Tapping lightly with the finger along the course of the nerve, midway between the zygoma and the angle of the mouth, results in a contraction of the ala of the nostril, angle of the mouth, and in most cases the inner canthus of the eye and the eyebrows. The appearance of a Chvostek phenomenon under 2 years, in the absence of birth trauma, indicates tetany. After 3 years the Chvostek phenomenon is not infrequently found in milder grades, in apparently normal children.

Trousseau's sign, elicited by moderately compressing the nerves and vessels of the arm midway between the elbow and shoulder by the hand or an elastic constrictor, is evidenced when present by the development of a carpal spasm (obstetrical hand).

Active Signs. Carpopedal spasms or arthrogryposis

Fig. 20 Spasmophilia. Trousseau's sign. (Bilateral.)

(state of tetany), are seen clinically as tonic spasms of the hands and feet. The fingers are usually flexed at the metacarpo-phalangeal joints and the phalanges extended; the thumbs are adducted almost to the little finger; the wrist is flexed at an acute angle, and the whole hand drawn somewhat to the ulnar side. If the spasm is very marked no motion is allowed at the wrist, but movements at the elbow and shoulder are usually

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normal. The feet are strongly extended, sometimes in the position of typical equino-varus and the first phalanges of the toes are flexed. If these conditions persist for a long time, edema of the dorsal surfaces of both hands and feet will develop. The clonic contractions at times remain for hours, and even days, and are evidently quite painful.

Convulsions (Eclampsia). Evidenced by loss of consciousness, spasms of the face and extremities at first tonic, later clonic. They usually last only a few minutes—may or may not recur. In exceptional cases a "status eclampticus" develops. More commonly, however, in the milder types the infant will recover from the individual attack quickly and without seeming after-effects, thereby resembling the petit-mal attacks of epilepsy. Only in the severest forms do they resemble the grand-mal attacks and only rarely is coma seen following the seizure. Due to the fact that spasmophilia is most frequently seen during the active period of early dentition and that many of the cases show delayed dentition the laity are inclined to ascribe teething erroneously as a cause.

Laryngismus Stridulus. (Inspiratory laryngospasm.) May occur without provocation or following crying or fright. There is an inspiratory crow, due to spasmodic closure of the larynx, associated with cyanosis and it may be followed by convulsions. The condition may persist over a period of several weeks or until proper treatment is instituted.

Spastic apnea or *expiratory apnea* are usually described as "holding breath spells" and are usually mild and transitory. They may result in cardiac death.

Course. The acute symptoms may last from a few days to several weeks. It varies in most cases directly with the treatment. Occasionally a persistent type is met with. Rickets, nutritional disturbances and infections must be overcome.

Prognosis. *Acute Attack.* It should always· be guarded, as it depends upon the underlying conditions which account for the symptoms. Convulsions should always be considered as symptoms of grave importance. The presence of thymus enlargement has a direct influence on the mortality rate.

Pertussis should be considered a serious complication. In the majority of cases the prognosis is, on the whole, good.

After-life. While many of these children show no after-effects, a considerable number suffer from nervous manifestations in later life, as headaches, pavor nocturnis, enuresis, tic, stuttering, etc. Others show mental retardation.

Treatment. *Dietetic. Latent spasmophilia* should be treated prophylactically to prevent the development of the manifest symptoms. As spasmophilia is relatively rare in *breast-fed* infants, the latent spasmophilic should, by preference, be fed human milk either from its mother or a wet-nurse. In cases in which the condition does develop in breast-fed babies, it often disappears if the infant is given breast milk from another source or a mixed diet. When this is impossible the cow's milk should be limited to a pint a day or it may be replaced by albumin (eiweiss) milk which has a low whey content. A cereal and vegetable diet should be instituted whenever the infant's age permits. Fruit juices are also essential. It is often well to keep the diet as small in quantity as is compatible with progress in the child. A temporary stationary weight should not be considered as cause for concern. Sodium and potassium salts should be avoided. A certain group of *artificially fed* infants will do better when cow's milk, in all forms, is greatly reduced. If the latent phenomena recur when cow's milk is again increased, these infants should be fed human milk for a considerable time. In such cases a mixed carbohydrate diet, consisting of cereals and sugars, to-

gether with vegetable purées and fruit juices, should
be instituted whenever possible.

They should not be kept for too long a time on a
strict cereal diet, due to the danger of development of
"flour injury."

Medicinal. Codliver oil alone, or combined with phos-
phorus, offers the best form of medicinal treatment
during the latent stage, however in the presence of ac-
tive manifestations they must be supplemented by other
forms of treatment.
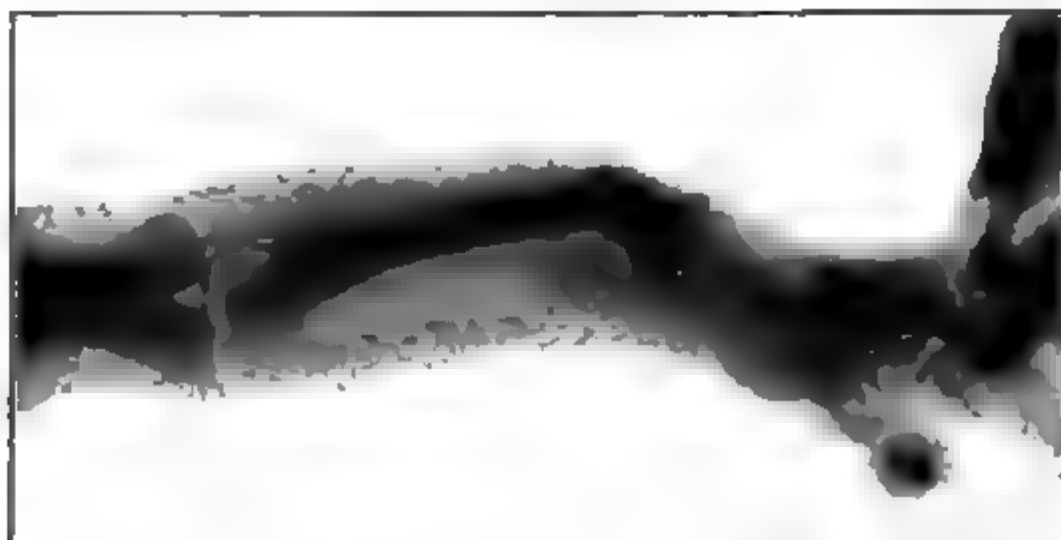
Fig. 21.—Spasmophilia—Pathological fractures of radius
and ulna with angular deformities following carpal spasm.
(Bilateral.)

The calcium salts in the form of chloride, lactate or
bromide, may be used to advantage both during the
active and latent stages. The calcium salts should al-
ways be prescribed in solution to prevent injury to the
mucous membrane. Calcium lactate or calcium chloride
may be administered in 0.5-1. Gm., three or four times
daily, or calcium bromide in one-half these amounts.
They are best administered in the food mixtures.

Convulsions. An initial dose of castor oil or magma
magnesia is a valuable adjunct to the further treatment
in the absence of marked gastro-intestinal irritation. A
short period of starvation diet, consisting of tea and

saccharine, should be followed by human milk where possible. When it is necessary to feed artificially the infant should be kept for twenty-four hours following the short starvation period on cereal gruel plus sugar, this to be followed by low milk and carbohydrate meals or albumen milk. Following this the previously recommended dietetic treatment may be instituted. It may be necessary to control the active manifestations by bromides per mouth and chloral hydrate per rectum, and when the temperature is high saline enemata, packs, sponging and baths are indicated. In the presence of repeated convulsions we have frequently seen excellent results following the administration of 10 cubic centimeters of an 8 per cent. solution of magnesium sulphate, hypodermically daily for one, two, or three days.

Further treatment should include the secondary anemia and not infrequently a change of climate is of advantage so that an outdoor life can be lived. Under all circumstances a good hygienic and dietetic regime should be instituted. Heliotherapy is of especial importance in all cases developing in the course of rickets.

Efforts should be directed toward the prevention of all respiratory infections, more especially during the winter and spring months.

Parathyroid feeding has shown no results.

PART VII.

Scurvy.

Scurvy is primarily a deficiency disease, due to insufficiency of the antiscorbutic vitamine. The disease follows improper diet. The chief pathological changes are noted in the blood vessels and bones. There are two distinct clinical types: the *acute* and the *subacute*. The *acute florid* type presents the classical picture of malnutrition, secondary anemia and hyperesthesia. Skin, mucous and serous membrane, visceral and subperiosteal hemorrhages develop. *Subacute* or *latent* scurvy is the most common form of the disorder and is characterized by an insidious onset of weeks or months, evidenced by lack of gain or loss in weight, anorexia, irritability and secondary anemia. Later, hemorrhages may occur. This type is more frequently overlooked, as it does not manifest pronounced symptoms. The general improvement of these symptoms following antiscorbutic treatment confirms the diagnosis.

Etiology. Congenital and inherited disease apparently play no part as a causative factor. It occurs most frequently between the ages of 6 and 15 months, half of the cases occurring between the seventh and tenth months, although occasional cases have been reported in babies under one month old. The age of development of the disease depends upon the length of time that a diet containing an insufficient amount of antiscorbutic has been fed; *even in a decidedly deficient diet a considerable time is necessary before the disease becomes clinically manifest.* The age at which it most frequently

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appears is that in which artificial feeding on cow's milk and dextrinized cereal diet, or other diets low in antiscorbutics, are most commonly required. This age might be referred to as the period of limited diet. Spontaneous cure in undiagnosed cases is the rule when the infants reach the age of fresh cereal, fruit and vegetable diet.

Scurvy is rare in the *breast-fed* infants, but does occur. The health and especially the diet of the mother are important predisposing factors. McCollum and Simmonds[1] have shown that in lactating rats the various vitamines cannot be synthesized and the content in the milk depends largely upon the amount eaten with the food. Hart, Steenbock and Ellis,[2] and Hess, Unger and Supplee,[3] have also shown this to be true of cows fed upon a diet deficient in antiscorbutic vitamine. Even when the lactating mother receives a diet deficient in vitamines, the cellular substance of the mother's body contains some, which the catabolic processes liberate, so that the milk cannot be entirely lacking in vitamines although the content may be too small to prevent scurvy. Secondly, even though the mother's diet is rich in vitamines, if she produces only a small quantity of milk, the amount of antiscorbutic vitamine will naturally be insufficient and thus predispose to scurvy in the exclusively breast-fed. Rickets, other diseases affecting the infant's metabolism, and infection may all predispose to scurvy, in the breast-fed as well as in bottle-fed infants. Infection as a precipitating factor will be discussed more fully later.

Most of the scurvy cases develop in *artificially fed* infants. As in the breast-fed, the absence of some vital

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[1] McCollum, E. V. and Simmonds, N.: Amer. Jour. of Phys., 1918, 46, 275. McCollum, E. V., Simmonds, N. and Pitz, W.: Jour. Biol. Chem., 1916, 27, 33.

[2] Hart, E. B., Steenbock, H. and Ellis, N. R.: Jour. Biol. Chem., 1920, 42, 383.

[3] Hess, A., Unger, L. and Supplee: Jour. Biol. Chem., 1920, 48, 229.

element in the food is the causative factor. Starvation in itself will not cause the disease, providing enough antiscorbutic substance is given. The time factor is very important in that diets low in antiscorbutic content must be fed over a prolonged period to cause the disease. Scurvy occurs most frequently in infants fed on proprietary foods. These are composed largely of dextrinized cereals, they are subjected to a long process of heating and although milk is added, the quantities are insufficient to meet the antiscorbutic needs. In addition, the milk used is usually subjected to prolonged or excessive heating, and in large cities where pasteurization is compulsory, frequently to a second heating and consequent ageing. Scurvy is much rarer where milk is the principal article of diet. Cow's milk in itself is relatively low in antiscorbutic.[1][2] It is estimated that a pint of raw, fresh cow's milk daily contains enough antiscorbutic to protect an infant from scurvy, when the animal from which it is obtained has been on a well-balanced diet.[3] The winter diet of cattle which until the introduction of ensilage was composed largely of grains and hay, resulted in the production of a milk with a lessened antiscorbutic content. This may have been a predisposing factor in the reported seasonal tendency of the disease. High dilutions of milk with a lessened quantitative content is another factor; this is comparable to the scant lactation in the breast-fed.

Ageing appears to be a more important factor than the heating of milk as far as the reduction or destruction of the antiscorbutic element is concerned. Hess[4] states that boiled milk is less liable to induce scurvy

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[1] Moore, J. J. and Jackson, L.: Jour. A. M. A., 1916.

[2] Chick, H. and Hume, M.: Lancet, 1918, i, 1, Barnes and Hume, M.: Lancet, 1919, ii, 323. Cohen and Mendel: Jour. Biol. Chem., 1918, 35, 425.

[3] Hess, A., and Unger, L.: Amer. Jour. Dis. of Children, 1919, 17, 221.

[4] Hess, A.: Amer. Jour. Dis. of Children, 1917, 14, 337.

than is pasteurized milk, due, he believes, to the fact that the long-continued exposure to heat in pasteurization (145° F. for 30 minutes) has a more destructive action than a short boiling (212° F. for a few minutes). His conclusions lead one to believe that he considers two factors of prime importance: first, ageing in itself, and, second, ageing plus the added factor of heating to a definite degree. He considers the possibility of oxidation during the period of ageing as being the destructive factor.

Hess[1] further states that when fresh cow's milk is dried in a few seconds at a temperature of 240° F. (116° C.), little of its antiscorbutic property is lost if kept hermetically sealed to prevent oxidation. We have confirmed this by feeding guinea pigs dried milk prepared by the Just Hatmaker process.[2] It must, however, be fed in sufficient quantities, which is best accomplished by feeding a concentrated mixture. The exact value of dried milk as a complete diet for babies is open to question, in the light of our personal experiences.

While scurvy occurs occasionally in breast-fed infants and in those fed on raw cow's milk, it is seen more frequently in infants fed on boiled, pasteurized and condensed milk, and in these cases, as previously stated, two factors are important—prolonged heating and ageing of the foods, either before or after heating. The same factors are important in the heating of proprietary cereal foods plus limited quantities of cow's milk. In these diets the vitamines have been destroyed or made less active in the cereals and are insufficient in the milk to meet the needs of the infant. A predisposing constitutional factor in the individual is not to be overlooked, and probably accounts for the precipitation of the disease in some infants while others escape on the same diet.

[1] Hess, A., and Unger, L.: Jour. Biol. Chem., 1919, 38, 293.
[2] Moore, J. J., Hess, J. H., and Calvin, J.: Unpublished experiments.

Previous diseases in the artificially fed, such as rickets, marasmus and other disturbances of metabolism, probably play a rôle in predisposing to scurvy, because all of these conditions presuppose a poorly regulated diet, both quantitatively and qualitatively, which diet would probably also be unbalanced in its antiscorbutic element.

The relation of infections to scurvy has been widely discussed recently. Epidemics of scurvy have been recorded in babies' institutions. These probably were cases of latent or subacute scurvy, in which the acute florid type was precipitated by a superimposed parenteral infection. Further, experience has led us to believe that scurvy predisposes to intercurrent infection, more especially of the skin, respiratory and gastro-intestinal tracts. These secondary bacterial infections may cause hemorrhagic lesions and the symptoms of the intestinal and infectious disorders become so intertwined as to be indistinguishable.

Summarizing, we believe the evidence of a specific organism as a factor is absolutely inconclusive. However, parenteral infections may precipitate the development of scurvy, or be a factor in causing a latent case to become active. Enteral infections, by preventing normal metabolic exchange, may pave the way for the development of scurvy, as may also be the case in the absorption of abnormal intestinal substances which are the end-products of bacterial activity.

The relation of *vitamines* to these so-called deficiency diseases is a much discussed subject and much experimental work has been undertaken in this direction. Funk,[1] in 1911, first called attention to this relationship. He classed beriberi, scurvy, pellagra, and rickets as deficiency diseases.

The beading of the ribs, formerly thought so characteristic of rickets, may also occur in uncomplicated cases

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[1]Funk, C.: Lancet, London, 1911, ii, 1266. Die Vitamine, Wiesbaden, 1914.

of scurvy in infants, and very often the rosary quickly becomes less or disappears when orange juice or other antiscorbutic food is given. This beading has been observed in scorbutic guinea pigs by Moore and Jackson,[1] and others. (Fig. 22.) It is truly scorbutic, as it shows the various microscopic appearances of scurvy. Beading of the ribs may also come about as the result of a lack of the water-soluble vitamine, although less

Fig. 22.—Scurvy in guinea-pig showing beading at the costochondral junctions. (*Jackson* and *Moore.*)

frequently than in scurvy. This latter type of beading decreases in size when the water-soluble element is added to the diet.—Hess.[2] Andrews[3] states that in eighteen cases of beriberi he encountered three instances of beading of the ribs at necropsy, although he had never seen rickets at necropsy in the Philippines Darl-

[1] Moore, J. J and Jackson, L., Jour. A. M. A., 1916, 67, 1931.
[2] Hess, A and Unger, L. Jour A. M A 1920, 74, 217.
[3] Andrews, W. L. Philippine Jour. of Science, 1912, 7, 67.

ing[1] states that scurvy resembles beriberi in the nervous involvement, both having increased knee-jerks, a much increased cardio-respiratory rate, and general and optic nerve edema. Pathologically. both show enlargement of the heart and fatty degeneration of the heart muscle and vagus. Nichols[2] states that in the tropics adult cases of scurvy often resemble pellagra and that they have much symptomatology in common.

Classification and Distribution of Vitamines. McCollum and Davis[3] have demonstrated two vitamines, fatsoluble A and water-soluble B (antineuritic). Most investigators now agree that there is at least a third one, namely: water-soluble C (antiscorbutic). These substances are constituents of the cells of both animal and plant tissues, and the content runs parallel to the cellular element of the foodstuffs. Although all natural foodstuffs contain certain amounts of these indispensable components of the diet, there is a great variation in the relative and absolute amounts contained. Thus, the best sources of *fat-soluble* A are animal fats, egg yolk fats, fish oils, milk fat and the leaves of plants; *water-soluble* B in yeast, fruit juices, vegetables and grain embryos. The leafy vegetables and those growing above the ground, such as tomatoes and celery, contain it in larger proportions than the root vegetables, such as potatoes, carrots and turnips; *water-soluble* C, the antiscorbutic element, is found in practically all fresh animal and vegetable tissues and fruits but to a much greater extent in the latter. It is present in actively living cells, so that in general those vegetable tissues which contain relatively large numbers of actively respiring cells (leafy vegetables), are richer in antiscorbutic power than are the roots or tubers. This generalization is not without

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[1] Darling, S. T.: Jour. A. M. A., 1914, 63, 1290.

[2] Nichols, L.: Jour. of Tropical Med., 1919, 22, 21.

[3] McCollum, E. V., and Davis, N.: Jour. Biol. Chem., 1915, 23, 181.

exception. Different vegetables and fruits vary greatly in their antiscorbutic potency. They differ widely also in the extent to which their antiscorbutic value will deteriorate under certain physical and chemical conditions (drying, alkalinizing, etc.). From the above statement it is apparent that the antiscorbutic potency of foodstuffs varies directly with the quantity contained.

Value of Various Foodstuffs. Considerable research has been conducted in the past few years to determine the antiscorbutic value of various foodstuffs, and the effect of ageing, drying, heating and alkalinizing them.

Milk contains a moderate amount of all three vitamines. Milk alone is a complete food for a number of animal species, *i.e.*, rats and swine. However, guinea pigs suffer from scurvy on a diet of oats and milk, even when raw and fresh, according to McCollum[1] and Moore,[2] which is a difficult fact to explain on a vitamine hypothesis, except that the amount of milk which a guinea pig will drink of its own accord does not contain a sufficient amount of antiscorbutic to protect it from scurvy. Barnes and Hume[3] state that 100 to 150 cubic centimeters daily of raw cow's milk will prevent scurvy in a guinea pig, but that such a large daily intake of milk causes digestive disturbances. Hess[4] states that 80 cubic centimeters of fresh, raw cow's milk will prevent the appearance of scurvy in guinea pigs.

Many fruits are excellent antiscorbutics. Orange juice is one of the best antiscorbutic substances. It also contains the water-soluble B vitamine, which is essential for growth and thus markedly stimulates growth. It should be fed in considerable quantities, varying from 15 to 45 cubic centimeters daily for the latter effect.[5]

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[1] McCollum, E. V. and Pitz, W.: Jour. Biol. Chem., 1917, 31, 229.

[2] Moore, J. J. and Jackson, L.: Jour. Inf. Dis., 1916, 19, 478.

[3] Barnes, K. and Hume, M.: Lancet, London, 1919, 11, 323.

[4] Hess, A. and Unger, L.: *Ibid*.

[5] Byfield, A., and Daniels, A. L.: Am. Jour. Dis. of Children, 1920, 19, 349.

Orange juice is still efficient after ten minutes' boiling; it can be dried rapidly at a low temperature and yet contain a significant amount of antiscorbutic substance after three months.[1] This property is present in the alcoholic extract of the juice, but not in the residue.[2] According to Hess,[3] artificial orange juice will not act as an antiscorbutic. The same author found that orange juice may be filtered, boiled, rendered faintly alkaline and given intravenously, affecting a prompt cure of scurvy. Harden and Zilva[4] state that when orange juice is rendered slightly alkaline and allowed to stand several hours it retains only a trace of antiscorbutic. They conclude that alkalies probably markedly reduce the antiscorbutic property of food in a few hours. Lemon juice is about as effective as orange juice but not as readily taken by infants. Fresh limes have only one-fourth the power of fresh lemons, while preserved limes have no antiscorbutic power.[5] Grapes[6] have only about one-tenth the antiscorbutic power of oranges, and bananas and apples also are poor in it.[3] Prunes have no value as antiscorbutic.[2]

Raw, fresh tomatoes are very efficient antiscorbutics and in contrast to some of the other vegetables can be dried rapidly or canned without losing much of this potency. Hess[7] found that one to four ounces daily of strained canned tomatoes will protect an infant from scurvy. Tomatoes are also rich in the water-soluble B and the fat-soluble A vitamine, according to Osborne and Mendel.[8] Thus, this vegetable contains all three

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[1] Givens, M. H., and McClugage, H. B.: Am. Jour. Dis. of Children, 1919, 18, 30.

[2] Hess, A., and Unger, L.: Jour. Biol. Chem., 1918, 35, 479—487.

[3] Hess, A., and Unger, L.: Am. Jour. Dis. of Children, 1919, 17, 221.

[4] Harden, A., and Zilva, S. S.: Lancet, 1918, 11, 320.

[5] Chick, H.: Lancet, 1918, 11, 735.

[6] Chick, H., and Rhodes, M.: Lancet, 1918, 11, 774.

[7] Hess, A., and Unger, L.: Jour. Biol. Chem., 1919.

[8] Osborne and Mendel: Jour. Biol. Chem., 1919. Ibid, 1920.

vitamines. Canned tomatoes are a valuable antiscorbutic for institutional use, less practical in private practice.

Potatoes are not especially rich in this element but because of the large quantity consumed by the population, these tubers afford a protection against scurvy, although much smaller quantities of the more potent orange juice would suffice.

Specificity of Vitamines. Yeast, which contains the water-soluble B, which is a specific for beriberi, has practically no effect on scurvy or rickets, although it does stimulate growth.[1] Byfield[2] states that orange juice, when deprived of its water-soluble B, still prevents and cures scurvy, but does not stimulate growth, which it does, however, when water-soluble B is left intact, if large enough quantities are given (about 45 cubic centimeters a day). Although orange juice prevents and cures scurvy, it has practically no effect on rickets.[3] Cod-liver oil, which contains the fat-soluble vitamine in large amounts, prevents and cures rickets in the presence of a sufficient quantity of the phosphorus ion, but has no effect on scurvy or beriberi. McCollum and Pitz[4] showed that oats, when fed with fats and salts, were sufficient for proper growth and development in rats, and concluded that thus oats contained sufficient water-soluble vitamine. However, guinea pigs suffer from scurvy when fed only oats, which again shows that the absence of water-soluble vitamine is not responsible for scurvy.

Pathological Anatomy. Two theories have received recognition regarding the underlying factors influencing the development of the pathological changes.

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[1] Hess, A.: Am. Jour. Dis. of Children, 1917, 13, 98.

[2] Byfield, A. H., and Daniels, A.: Am. Jour. Dis. of Children, 1920, 19, 349.

[3] Mellanby, E.: Lancet, London, 1919, 1, 407.

[4] McCollum, E. V., Simmonds and Pitz, W.: Jour. Biol. Chem., 1917, 29.

The first regards the changes in the bone marrow as the primary factor with resulting interference with the function of the hematopoietic system.

The second suggests primary blood-vessel changes and classifies it essentially as a hemorrhagic disease, without important primary blood changes, and assumes that the same causative factors are responsible for the blood-vessel and bone marrow changes.

The most prominent pathological manifestations are

Fig. 23.—Scurvy showing lateral displacement of the metaphysis of both the upper and lower ends of the tibia and fibula. A similar displacement was present in practically all of the long bones. A subperiosteal hemorrhage is seen along the outer surface of the tibia which shows beginning calcification. In the process of healing in this infant that part of the metaphysis extending beyond the line of the shaft was completely absorbed in each instance and the bone took on a normal development in the direct line of the shaft. The "white line of Fraenkel" in this positive appears as a dark shadow, above this a rarified area is seen.

found at the seat of most active bone growth, as at epiphyseal ends of the shafts of the long bones, the ends of the ribs and the skull, due to changes in all of the

bone structures. A failure of the integrity of the epithelium of the blood-vessels is the underlying factor in the generalized hemorrhagic manifestations in the subperiosteal tissues, the gums, skin and the hematuria.

The gross pathological findings in a case of acute scurvy are characteristically typical, especially in the long bones. Grossly, there usually are large or small sub-
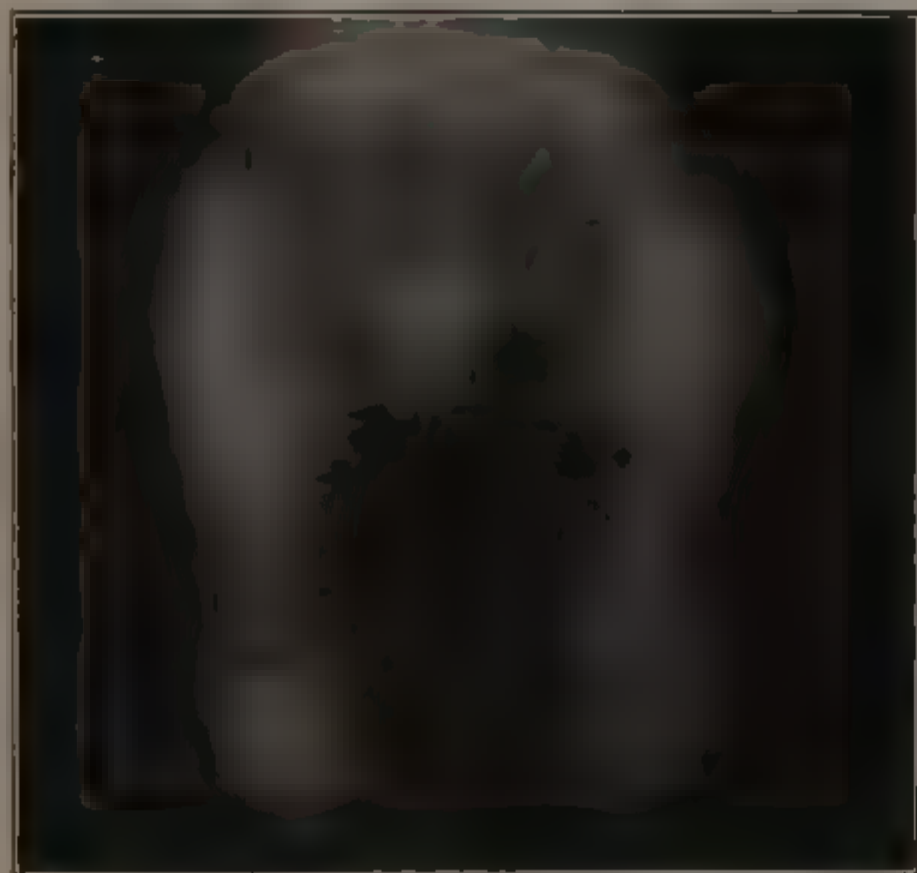
Fig 24.—Scurvy. Hemorrhage into both knee joints with displacement of left epiphyseal nucleus. Also showing white line.

periosteal hemorrhages, most frequently found along the shafts and distal ends of the long bones of the lower extremities. The periosteum of the long bones is congested and swollen. Inflammatory changes rarely occur and there is no small round cell or leucocytic infiltration. Extensive hemorrhage may invade the soft tissue following rupture of the periosteum. In old hemorrhages calcification of the elevated periosteum may be present

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and fractures are not uncommon. Due to the rarefaction of the cortex, transverse fractures either single or multiple may be present, due to muscle action, trauma, attempts at sitting, standing or falling. Lateral displacement of the metaphyses may result from trauma, torsion or hemorrhage. (Fig. 23.) Less frequently the epiphyses may be dislocated through intracapsular hemorrhage. (Fig. 24.)

Changes similar to those seen at the ends of the long bones are noted at the anterior ends of the ribs with resulting beading. Subperiosteal hemorrhages are not uncommon over the skull and more especially in the orbits resulting in exophthalmos. The latter may follow prolonged crying. The affection of the gums is due to the pressure exerted upon the mucous membrane by the teeth in dentition and also by external trauma as in biting. The latter more commonly results in hemorrhages in the upper jaw.

On sectioning the bone, the cortex is seen to be exceedingly thin and brittle, the trabeculæ being thin and reduced in number. The marrow at the ends of the long bones is yellow and gelatinous instead of red.

Microscopic changes, as seen in long bones, are most characteristically seen in the marrow. The bone marrow is poor in cellular elements, the connective tissue framework increases at the expense of the myeloid cells and blood-vessels. Hemorrhages are usually present in the marrow.

The thin and fragile bone and poorly developed trabeculæ are considered to be due to imperfect functioning of the osteoblasts, which are reduced in number and appear smaller than normal. There is not an excess of osteoclasts, consequently normal resorption of bone occurs with diminished regeneration.

The gross changes found in the heart in severe cases of scurvy usually consist in right-sided hypertrophy and fatty degeneration of the heart muscles. Occasionally

an increased quantity of fluid is noted in the pericardial sac. There are no characteristic lesions of the valves secondary to scurvy. Jackson and Moore[1] found a marked thinning of the walls of the blood-vessels in their experimental animals and not infrequently thrombosis of the veins was noted. No constant lesions of the blood vessels in human scurvy have been described. In all probability hemorrhage occurs by diapedesis. Hess[2] believes that a weakness of the vessel walls exists, as
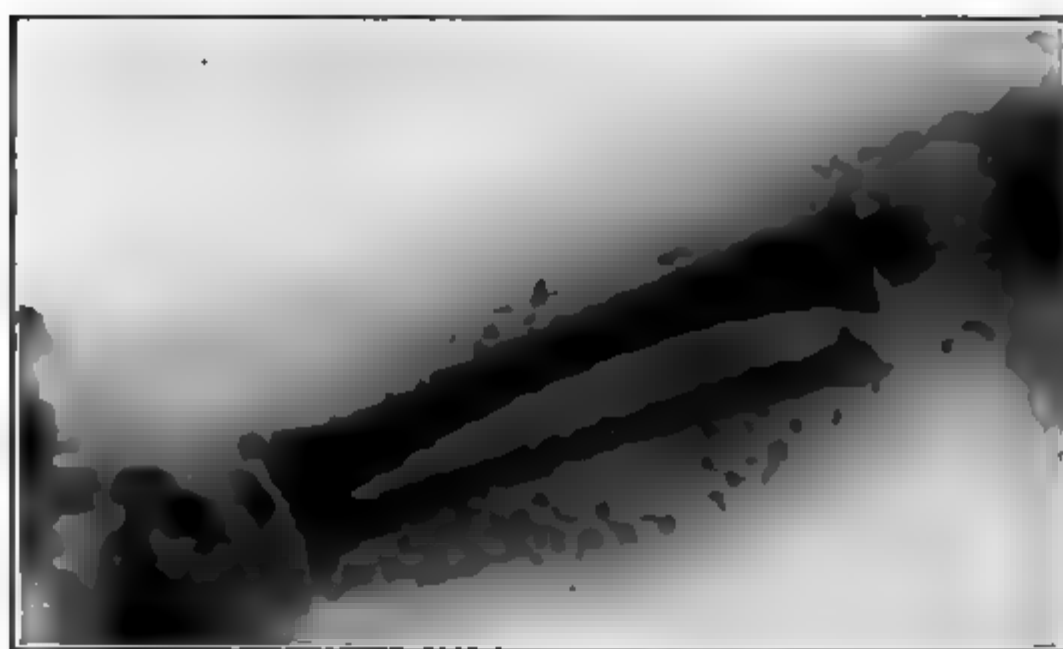
Fig. 25.—Scurvy. Showing fraying of the lower ends of the tibia and fibula. There is also a large hemorrhage about the tibia in this illustration whch casts a very hazy shadow. This hemorrhage is 24 hours old and followed trauma due to compression of the soft parts during the removal of blood from the heel for a Wassermann test.

demonstrated by the "capillary resistance test," which consists in applying a tourniquet to the upper arm, thus subjecting the vessel walls to additional strain, generally causing the appearance of numerous petechiæ on the forearm. Following the trauma caused in taking blood from the heel for a Wassermann test, a massive hemor-

[1] Jackson, L. and Moore, J. J.: Jour. Infect. Dis., 1916, 19, 478.
[2] Hess, A.: Jour. A. M. A., 1921, 76, 694.

rhage occurred into the soft tissues of the leg and foot in one of the author's cases. (Fig. 25.)

Hess and Fish,[1] studying the blood in scurvy, found the coagulation time to be normal or only slightly increased. No deficiency of blood platelets or calcium or excess of antithrombin could be demonstrated. During the late war, scorbutic patients operated upon showed no particular tendency to hemorrhages.

The respiratory tract usually shows no typical changes other than congestion and subserous hemorrhages. However, in some of the fatal cases secondary pneumonia, infarcts and a generalized edema may be present. The alimentary tract beside the swollen gums, which are usually the seat of more or less intense hemorrhage and not infrequently necrosis, shows no constant changes in the moderate types. In the severer forms hemorrhage may occur from any region of the stomach or the intestinal tract. The solitary follicles and Peyer's patches are usually infiltrated and may be ulcerated. The spleen is usually moderately enlarged and congested, as is also the liver. The latter may also be the seat of cloudy swelling and fatty degeneration; similar changes are usually present in the kidneys. The most frequent changes in the nervous system are seen in the peripheral nerves, hemorrhage into the nerve sheath being the most common. Hemorrhages are not uncommon in the brain substances and pachymeningitis hemorrhagica interna have been described repeatedly.

The lymph nodes draining areas of hemorrhage are enlarged. In guinea pigs McCarrison[2] noted frequent hemorrhages into the adrenals, greatly enlarging them, especially the medullary portion with degeneration of the cells in both cortex and medulla. The adrenalin content is much decreased. These findings have not been noted in human scurvy.

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[1] Hess, A., and Fish, M.: Amer. Jour. Dis. of Children, 1914, 8, 386.

[2] McCarrison, R.: Oxford University Press, New York, 1921.

Fig. 26.—Scurvy. Typical lesions of the gums, with hemorrhage at the free edge of the gums of the incisor teeth.

Symptoms. In the discussion of the symptoms of scurvy the acute and subacute types must be considered, and it must be remembered that the mild and severe forms of each of these will present varied clinical pictures. Due to the fact that the symptoms develop following a prolonged use of an insufficient diet, a secondary anemia, usually associated with more or less marked hemorrhages and bone changes, is found in infants showing other evidences of malnutrition. These may be the only clinical evidence of the disease in its mildest form. The typical acute case of severer type, after a more or less prolonged period of inanition, gives evidence of discomfort on being handled and shows a tendency to assume a flexed position of extremities. Tenderness on palpation of the limbs is noted and the infant shows a tendency to refrain from voluntary motion. These changes are most frequently noted in the lower extremities. Palpable and visible swelling along the shafts may develop, although they are not constant findings. When present they are the result of the subperiosteal hemorrhages. Less frequently these hemorrhages are noted in the epiphyseal regions but usually extend some distance along the shaft of the bone which tends to distinguish them from acute inflammatory joint conditions. Rare are the hemorrhages into the joints themselves, although they may occur. These may result in epiphyseal displacement. Among the rare lesions lateral displacement of the proliferating zones may exist. While swelling and discoloration of the gums is one of the most constant features, hemorrhage is not always visible. The typical lesions of the gums consist of a red or purple discoloration at the free edge of the gums of the incisor teeth. (Fig. 26.) This may exist on the posterior edge behind the teeth and not be present anteriorly. Hemorrhage in the peridental membrane, resulting in swollen and edematous gums, may be present before eruption of the teeth. Very rarely the gums may be

markedly affected when there is no other obvious symptom of scurvy excepting malnutrition and secondary anemia. Not infrequently the incisor teeth become loosened and are lost. Palatal hemorrhage, most frequently seen over the middle part of the vault of the hard palate, should be looked for. Petechial and ecchymotic hemorrhages may occur spontaneously in the skin but are more exceptional findings in the milder types unless secondary to trauma; more frequent are the orbital hemorrhages resulting in a discoloration about the eye and hemorrhages into the conjunctiva. Occasionally a proptosis is noted. (Fig. 27.) Discoloration of the upper lid is more frequent than in the lower. Epistaxis occurs often. Edema is occasionally noted over the thickened part of the limbs and about the face. The visceral mucous membranes are commonly the seat of hemorrhage, resulting in hemorrhages from the gastro-intestinal tract and the genito-urinary tract. The appearance of blood in the vomitus and feces varies with its source and the time that it has been in contact with the digestive juices. The urine is usually diminished in volume, may contain albumin, casts, red and white blood cells in the presence of hemorrhage. An early appearance of blood stained urine is not uncommon. Pus cells may be present in considerable numbers after the subsidence of the hemorrhage. Pyrexia in infantile scurvy, although rather the exception than the rule, is by no means a rarity. The temperature may be raised to 101° or 102° F. when the infant comes under treatment, but it subsides generally within a few days as the acute symptoms of scurvy disappear. Hess describes the cardio-respiratory syndrome, consisting of increased pulse and heart beat often over 150, with respirations reaching 50 to 60. He believes that these phenomena may be associated with a disturbance of the vagus mechanism. Further changes in the nervous system may be present, due to hemorrhage or focal degeneration.

The paresthesias due to peripheral nerve involvement have been described. Increased deep reflexes are usually present. The diagnosis of the subacute type is not always possible by the clinical findings and may be dependent upon the reaction to antiscorbutic diets. The possibility of its existence should always be considered in the presence of an insufficient or improper diet and when infants are fed on heated milk, which is over long periods without the addition of antiscorbutics to their

Fig. 27.—Scurvy. Bilateral proptosis, with discoloration about the eyes and hemorrhages into the conjunctivæ.

diet. Such infants usually show evidence of malnutrition and anemia, loss of appetite, increased tendon reflexes, irritability and rapid pulse and respiration. In this class of cases, unless the diet is corrected, the typical clinical picture may be precipitated at any time if the diagnosis is correct.

Radiological Diagnosis. The diagnosis of the disease in advanced cases may be made by the use of radiograms. The density of the subepiphyseal region is materially diminished where the lack of bone formation is especially marked. Just above this area, traversing the shaft in its transverse diameter, an irregular zone of

increased density is seen in advanced cases of scurvy. This is seen as a lighter zone in the *radiographic negatives* and is described as the "white line of Fraenkel." This shadow can especially well be seen on the lower end of the femur and the distal ends of the bones of the forearm. This shadow receives its explanation from the anatomical finding in this portion of the diaphysis of an irregular chaos of calcified trabeculae, calcium and lacy detritus, and also of bone marrow mixed with masses of blood and pigment. The structure of the spongiosa, which normally extends down to the place of transition of the shaft into the epiphysis, becomes suddenly broken and is replaced by a tissue, thickened by compression, and showing no more an orderly structure of trabeculae, and this abnormal tissue shows a corresponding shadow appearing always in the same position on the radiogram. From the above discussion it becomes clear that a certain degree of alterations in the bone is prerequisite for the appearance of a shadow in the radiogram. This is known as the "white line of Fraenkel." (See Figs. 24 and 25.) If this degree has not been reached, then the shadow is absent, even though the disease be present. A similar shadow is not infrequently seen in severe cases of rickets. Less constant than this shadow are the subperiosteal shadows at the seat of hemorrhages. (See Figs. 23 and 25.) These shadows may be invisible, even at the height of hemorrhage, becoming more and more distinct with age, due to the deposit of osteophytes. Fractures and infractions are demonstrable in scurvy. Other findings in scurvy are epiphyseal separation and displacements, hemorrhages within the joint capsule and occasionally, but rarely, intramuscular hemorrhages may be demonstrated.

Diagnosis. In the differential diagnosis of scurvy the possibility of its existence and its characteristic symptoms must constantly be borne in mind. The presence of tenderness over the bones and tendency to fixation

of the joints, the usual presence of multiple joint involvement, with evidence of hemorrhage in other parts of the body, in infants usually between 6 and 18 months of age and the history of improper diet or prolonged lactation, should lead to the suspicion of the presence of scurvy.

Rachitis. When marked rickets is present the recognition of a superimposed scurvy is often difficult and a careful clinical study of the case may be necessary for its recognition, unless the complicating symptoms are pronounced. The dietetic test is the best means of differentiation in the moderate types.

Rheumatic Fever. Pain, tenderness and swelling of the limbs in infants under 18 months should always lead to the suggestion of scurvy. In rheumatic fever the tenderness is usually associated with the joints and localized, while in scurvy there is almost invariably shaft involvement, even in the presence of joint involvement. The feeding history, presence of hemorrhages into the skin and from the mucous membranes will often complete the diagnosis.

Syphilis. Syphilitic osteochondritis is usually associated with a history of early lesions—most commonly it occurs before the sixth month and shows the characteristic blood and spinal fluid findings in the infant and parents. The painful swellings are usually seen about the ends of the long bones and the radiological findings are characteristic. In later manifestations, in which periostitis ossificans is a very important finding, we see newly formed subperiosteal masses with bony structure. They are usually most marked in the middle of the diaphysis of the tibia. Old cortex may often be differentiated from the newly formed subperiosteal bony masses. Cortical thickening is, therefore, an important point in differential diagnosis.

Purpura. This disease is characterized by hemorrhage, particularly into the skin. It is very rare in in-

fants. It can usually be excluded by the tenderness of the shafts of the long bones in scurvy. The roentgenological findings of scurvy will be absent. The intestinal and skin hemorrhages in the late stages of athrepsia may lead to some confusion unless a careful history is taken.

Osteomyelitis. Scurvy is frequently mistaken for osteomyelitis by those who forget the possibility of the former. Osteomyelitis is usually confined to one limb, is characterized by a continued high febrile reaction, and by leucocytosis, and lacks the tendency to hemorrhages in other parts of the body. The diagnosis can be settled by X-ray examination.

Simple Fractures and Dislocations. These can usually be differentiated by the history of trauma, the singleness of the lesion and the absence of other typical clinical and radiographic lesions.

Tuberculous Bone and Joint Lesions. The radiographic findings are usually sufficient for the differentiation of these two conditions.

Bone Tumors. The malignant types are characterized by severe systemic reaction, the usual finding of a single bone lesion and the tendency to metastases in the visceral organs.

Poliomyelitis. The pseudo-paralysis of scurvy may be mistaken for this condition. In the differentiation the history of an acute febrile onset in an infant previously in good health and the tendency to general tenderness of extremities, rather than localized, must be considered. The further progress of the case will usually complete the diagnosis.

Acute Nephritis. In the presence of edema and the urine containing albumin, casts and blood, the differentiation may be very difficult and it is not to be forgotten that acute nephritis may be present as a complication of scurvy. The course of the disease and the reaction to dietetic treatment may be the only means of differentiation, as to the importance of the urinary findings.

Pleuritic and Peritonitic Pain. A careful study of the chest and abdominal viscera for evidence of acute inflammatory conditions are necessary to eliminate the possibility of a confusion in the diagnosis between inflammatory changes in these parts.

All other conditions associated with bone and blood changes, and in the latter leading to hemorrhage, may at times require differentiation.

Prognosis. There are few diseases in which the effect of treatment is so striking as in *mild* infantile scurvy; under efficient antiscorbutic diet the tenderness and pain on movement are usually appreciably less in forty-eight hours; although such rapid diminution of the tenderness and pain may be expected, other symptoms persist longer; the subperiosteal hemorrhages, if large, may cause some deep thickening to remain for two or three weeks. Occasionally the thickening remains for many weeks or months, in spite of the complete disappearance of all other symptoms of scurvy. It should be remembered that an individual tendency to scurvy may exist and that further indiscretions in diet may readily lead to recurrences. In severe cases the prognosis must be guarded because of the liability to severe hemorrhages, syncope from the profound anemia, and death from diarrhea, broncho-pneumonia or exhaustion.

Treatment. *Prophylaxis.* In discussing the etiology, certain facts were mentioned, which it is of importance to bear in mind in a consideration of the prophylactic treatment of scurvy, namely: that this disease is the result of the deficiency or absence of a certain substance in the diet, which in all probability belongs to the so-called group of vitamines. Human milk usually contains a sufficient amount of this antiscorbutic to protect the infant, fresh, raw cow's milk, although relatively low in it, contains enough for the normal development of the infant if given in sufficient quantities, although its

content varies with the diet of the cow. This anti-scorbutic is reduced or destroyed by the ageing of milk; heating the milk has the same influence. Proprietary foods and cereals are especially prone to be deficient foods because of the process of heating in their preparation and the low vitamine content of the cereals. Many fruit juices and certain vegetables, especially if fresh, are rich in antiscorbutic. With this knowledge at hand, scurvy can nearly always be prevented by proper attention to the antiscorbutic value of the diet in infant feeding. If the diet is low in antiscorbutic, it should be supplemented by sufficient quantities of foods known to be rich in this substance.

If babies are breast-fed, they rarely develop scorbutus, so that breast feeding is one of the best preventive measures. To avoid the danger of the vitamine content of the milk becoming too low, the lactating mother should partake of a liberal amount of cow's milk, leafy vegetables and fruits. Moreover, when the mother's milk is scanty and so quantitatively poor in antiscorbutic, sufficient cow's milk, vegetables and orange juice should be added to the infant's diet to fulfill the baby's vitamine requirements. In the artificially fed, sufficient quantities of fresh, raw cow's milk will usually prevent scurvy, but the great danger of bacterial infection from contaminated raw milk usually more than counterbalances the danger of this disease. Consequently, unless certified milk can be obtained, it is better to boil the milk, even if previously commercially pasteurized, and during the very hot weather we even advise boiling certified milk. If, however, fresh raw milk is used, too dilute a modification should not be given, as here again the quantitative amount of antiscorbutic may be insufficient. This milk should be obtained, if possible, from cows properly fed on plenty of greens. During the winter, when the diet of the cattle is mostly grains, even the very young infants should be given orange juice.

The number of babies fed on heated milk, either pasteurized or boiled, is continually increasing. An antiscorbutic, such as orange juice, lemon juice, tomatoes, etc., should ·in every instance be given to these infants. We start as early as the sixth week with five drops diluted with water, twice daily, and gradually increased until two to four drams are given daily by the third month and about one ounce by the fifth or sixth month, so as to prevent the vitamine deficiency for even a short period and thereby avoid the danger of latent scurvy. If the babies object to it, it may be diluted with water and a little cane sugar added. If it is regurgitated, a small amount of alkali may be added just before feeding and then it is usually better tolerated. Orange juice is continued throughout early childhood. Strained, warmed (not cooked) tomatoes, either raw or canned, may be used instead of orange juice, in doses of one dram to one ounce daily. (The latter may be given to infants over three months of age.) These are as well borne as orange juice, even by very young infants. Later on, potatoes cooked with their jackets on serve as efficient antiscorbutics, as do also the leafy vegetables. As cooked vegetables have lost considerable of their antiscorbutic potency, the amount fed must be sufficient to protect against scurvy. One tablespoonful a day is usually adequate. The vegetables should be as fresh and young as possible.

Proprietary and other foods subjected to prolonged heating should be avoided as much as possible and if given must be supplemented by sufficient quantities of antiscorbutics.

The basis of all infants' diets should be fresh, clean milk.

Active Therapy. When scurvy develops, the active treatment is mainly dietetic, no medicinal agent influencing the underlying condition. Orange juice must be given at once, starting with small doses of a few drams

daily and rapidly increasing within two days or so to one or two ounces. This may all be given at one time, an hour before the mid-morning feeding, or divided into two or three doses. Exceptionally, in extremely severe cases, when orange juice cannot be given by mouth, it may be boiled, filtered and rendered faintly alkaline, and administered intravenously. Lemon juice is just as efficient but less palatable. Orange juice will cure scurvy, even if the causative food is continued, because the fruit juice supplies the lacking element in the diet. It is better to change the food, however, unless there is some special indication for continuing it. Fresh milk, even if boiled, should be added in sufficient quantities if the diet contains too little milk. Fresh green vegetables or vegetable soup may be added to the diet if the child is old enough to digest them. A tablespoonful daily of boiled, mashed potato is a good antiscorbutic. Other vegetables, as tomatoes, carrots, celery, turnips, and green leaves, are good antiscorbutics but their efficacy is much below orange juice. They should be fed when the infant's age permits. (See additional foods, page 161.)

Even in the acute florid type of scurvy, recovery is rapid and complete under the above treatment. Tenderness of the extremities disappears in a few days and the hematuria usually ceases within a week. The swelling lasts much longer. The dietetic treatment should be continued for a period of months so that bones and tissues may be fully restored to normal.

Actively scorbutic infants should be moved and handled as little as possible for the first few days under treatment. Sedatives may be necessary for the first day or so to control the pain. If fractures or epiphyseal dislocations occur, splints are necessary for immobilization. In cases of marked anemia and severe hemorrhages, a blood transfusion may help. During convalescence, iron preparations should be given.

Rachitis, if present, should be recognized and treated simultaneously.

Good hygienic surroundings should be provided to aid in convalescence.

PART VIII.

Acidosis.

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By the term acidosis we refer to that condition in which there is a diminution in the reserve supply of fixed bases in the body fluids and tissues. The physio-chemical reaction of the blood remains unchanged except in extreme conditions.

Acidosis may result from any one of the following factors or a combination of them:

(1) An insufficient intake of food and fluid, with re-sulting dehydration, or excessive production of acetone bodies, or both. (Anorexia, starvation, vomiting and diarrhea.)

(2) Incomplete oxidation of the organic acid radicals. (Diabetes, cyclic vomiting; infections, anhydremia, and conditions resulting in insufficient aëration of the blood as cardiac and pulmonary disease, suffo-cation, excessive exercise, etc.)

3) Defective neutralization of acid in the body by am-monia. (Nephritis.) This is due to the fact that the acid phosphates causing the acidosis fail to call forth ammonia production rather than the failure of ammonia production *per se*.

(4) Inadequate elimination:

a. Of carbon dioxide by the lungs. (Pulmonary disease.)

b. Of acid by the kidneys. (As acid phosphates in nephritis and anhydremia.)

A sharp distinction must be drawn between aceto-nuria and acidosis. This is necessary because of the wide-spread confusion which has arisen, in the interpre-tation of the relationship which acetonuria bears to acid-

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osis. Acetonuria consists merely in the presence of acetone-bodies in the urine, and may or may not be accompanied by symptoms of acidosis. Acetone body *acidosis* should be applied to that condition in which an excess of these bodies are present in sufficient quantity to neutralize enough of the bases to appreciably diminish the alkali reserve. Acidosis may, however, be due to an excess of other acid-bodies, such as acid phosphates, lactic acid and carbonic acid.

Mechanism of Acid-base Equilibrium. As the blood plasma has approximately the same composition as the tissue juices and the relative alkalinity of the tissues has a fairly constant relation to the alkalinity of the plasma, it is sufficient to study the regulation of the acid-base equilibrium in the blood. Acids are constantly being produced in the course of metabolism. Protection is afforded the body against these in fundamentally the same manner in health and disease. In the latter the body responds within its reserve capacity by a quantitative increase in its normal processes of oxidation, excretion and neutralization.

The blood is normally slightly alkaline. Nevertheless, by virtue of its carbonates, phosphates and proteins, it can neutralize a moderate amount of acid. This ability to neutralize acid or base is termed "titratable value" in distinction from the actual reaction. The true or actual reaction of a fluid concerns itself only with that portion of acid or base which, after going into solution, becomes dissociated into hydrogen and hydroxyl ions. The titration value of a solution is a broader term, since it includes not only the free hydrogen and hydroxyl ions present, but all the reserve of undissolved and undissociated hydrogen or hydroxyl, which can be liberated as ions.—Sellards.[1] Henderson[2] has pointed out and

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[1] Sellards, A. W.: Princ. of Infant Feeding, Harvard U. Press, 1917.

[2] Henderson, L. J.: Amer. Jour. of Physiology, Vol. 21, 1908, p. 427.

established that the mono- and di- sodium phosphates and carbonates of the blood constitute one of the fundamental features in the regulation of neutrality, since the monosodium salt (NaH_2PO_4) is acid and the disodium salt (Na_2HPO_4) is alkaline. The normal carbonate and bicarbonate of the serum act in a similar manner and are present in much larger quantities and therefore of even greater importance. This mixture of phosphates and carbonates is the most effective in maintaining a constant reaction; it is extremely efficient not only in aqueous solution but in the presence of proteins, such as serum affords, and these moderately enhance its value. This action is practically limited to the hemoglobin, the other blood proteins having very little action in retaining the constant reaction of the blood, on account of the fact that they are inefficient in the particular reaction of the blood although on other reactions they have a marked effect.[1] Free carbonic acid is present in the body fluids in such concentration that it automatically converts into bicarbonate all bases not bound by other acids. The bicarbonate, therefore, under the conditions existing represents the excess of base which is left after all the non-volatile acids have been neutralized and is available for the immediate neutralization of further acids. In this sense it constitutes the *alkaline reserve of the body.*—Van Slyke.[2] The bicarbonate concentration of the blood is representative of that of the body fluids in general, and is normally maintained at a definite level, which forms approximately a 0.3 per cent. solution in the blood plasma. Entrance of free acids reduces it to an extent proportional to the amount of the invading acid. Sodium bicarbonate is the chief substance concerned with this mechanism.[3] The proteins and phosphates in the blood play a lesser rôle. An acid or even a neutral reaction of the blood is incompatible

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[1] Henderson, Y.: Jour. Biol. Chem., 1920.

[2] Van Slyke, D. D.: Jour. Biol. Chem., 1917, Vol. 30, p. 289.

[3] Howland and Marriott: Penna. Med. Jour., Vol. 21, 1918, p. 429.

with life, so that the phrase "increased acidity" really, means reduced alkalinity.

Carbonic, phosphoric, sulphuric and among the organic lactic acids are normally formed as end products in the metabolic processes. These acid substances must be neu- tralized and excreted without disturbing the normal de- gree of alkalinity of thé tissues if the organism is to remain in a state of health. When there is an inability on the part of the body to maintain its alkali reserve,. acidosis results.

The defenses of the body against acidosis are princi- pally pulmonary ventilation, excretion of acids by the renal tissues and neutralization of acids by ammonia.

Pulmonary Ventilation. In the blood carbonic acid in part unites with the alkalies in the blood to form bi- carbonate and some remains in the solution in the plasma. The venous blood thus loaded with carbonic acid is car- ried to the capillaries of the lung tissue. Here carbon dioxide diffuses through the thin capillary and alveolar walls into the alveolar air because the pressure of CO_2 is less in the inspired air than in the blood. Thus, car- bon dioxide is given off by the lungs without the loss of alkalies from the body and the blood becomes more alkaline again. If an excess of carbonic acid accumu- lates in the blood, even a very small increase of the H ion concentration stimulates the respiratory center, with a resulting increased rapidity and depth of respira- tion and increased CO_2 excretion. Sellards has a some- what different explanation for this hyperpnea. He main- tains that as the fixed bases become depleted the oxygen is still carried by the arterial blood but the resulting carbon dioxide gradually accumulates in the tissues,: since there is not an adequate supply of bases for trans- porting it to the lungs. With this accumulation of car- bon dioxide, the tissues are no longer able to utilize the oxygen brought to them; the individual becomes dys- pneic and suffers from subjective symptoms of suffo-

cation just as though he were deprived of air. The oxygen, since it cannot be taken up by the tissues, remains in the venous blood, giving it an arterial color.

Howland and Marriott[1] state: If, on the other hand, an acid, such as beta-oxybutyric or lactic, is poured into the plasma, as it passes through the tissues, a certain amount of the bicarbonate is neutralized and carbon dioxide is set free in solution. The excess of carbon dioxide is removed as before and the blood reaction returns to normal. Some bicarbonate has, however, been neutralized so that the plasma has become a weaker solution of bicarbonate. When this solution takes up carbon dioxide from the tissues its reaction becomes more acid with the same amount of dissolved carbon dioxide as formerly. Roughly speaking, if the bicarbonate of the plasma is reduced one-half, only half as much carbonic acid can be carried in solution as formerly, if the reaction is to remain the same as before. By doubling the ventilation in the pulmonary alveoli the dissolved carbonic acid of the plasma may be diminished about one-half; at the same time the concentration or tension of carbon dioxide in the pulmonary alveoli is also diminished one-half. The carbon dioxide tension in the alveolar air, for this reason, is a measure of the extent to which the bicarbonate reserve of the plasma is depleted, and increased pulmonary ventilation is one of the symptoms of acidosis.

Increased pulmonary ventilation serves to prevent a significant change in the reaction of the blood, but it cannot prevent a depletion of the bicarbonate reserve of the plasma. This reserve eventually would be entirely exhausted were it not for other means of defense and for the provision for a replenishment of the alkali reserve.

Excretion of Acid by Renal Tissues. An acid urine is excreted normally by the kidneys from an alkaline

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[1] Howland and Marriott: *Ibid.*

blood, thus sparing alkalies to neutralize more acids of metabolism. A small amount of free acid is present in the urine. Most of the acid in the urine, however, is excreted as acid phosphate which is formed from the alkali phosphate of the blood plasma, by the following reaction: Na_2HPO_4 plus H_2O plus CO_2, equals NaH_2PO_4 (excreted in urine) plus $HNaCO_3$ (remains in the blood). The base thus spared serves to replenish the bicarbonate reserve. Some alkaline phosphate is, however, excreted in the urine, and although no alkali is lost through the lungs, some is lost in the urine by the excretion of the salts of phosphoric, sulphuric, lactic acid, etc. To compensate for this alkali loss there is usually a sufficient quantity of alkalies taken with the food.

Neutralization of Acids by Ammonia. Ammonia formed instead of neutral urea, neutralizes acids in the plasma and is excreted in the urine as ammonium salts. Ammonia occurs in the urine physiologically only to the extent which is needed for the neutralization of acids, the remainder of the ammonia being promptly converted into urea. When there is an increased accumulation of acids in the blood, more ammonia is formed to neutralize them and is an important safety factor in maintaining the alkali reserve. The presence of an increased amount of ammonia in the urine does not, in itself, indicate an acidosis but rather that the body is reacting to prevent acidosis.

Clinical Types. As previously stated, it is necessary to draw a distinct line between acetonuria and acidosis. Most of the studies of the past have dealt mainly with the acetone body acidosis. However, in the light of more recent experimental and clinical investigations the prominence of other acid bodies such as the acid phosphates, carbonic and lactic acid have assumed a more prominent position as causative factors, either as distinct or mixed types of acidosis.

Acetone Body Acidosis. Acidosis in the course of diabetes is a recognized representative of this type. The development of hyperpnea and drowsiness, in the course of diabetes, indicates clinically that such an acidosis exists. A study of the CO_2 tension in the alveolar air and the bicarbonate reserve of the plasma should always be made with the first clinical or laboratory evidence of a beginning acidosis. As a prophylactic measure such tests should be made at regular intervals during the course of the disease. Fortunately, diabetes is a rare disease in children, but a very fatal one. Acidosis of this type occurs in the presence of ileo-colitis (bacillary dysentery), also at times at the onset of measles, scarlet fever, and in the presence of respiratory infections. Other conditions, such as recurrent vomiting, acute infections and nutritional disturbances which are commonly associated with acetone body acidosis will be treated individually because of their clinical importance.

Acidosis in the Course of Acute Infectious Diseases. Acute toxic conditions out of proportion to the seeming severity of the infection are frequently seen in the course of acute infectious diseases in young children. They are frequently due to the production of sufficient amounts of acid bodies to cause a definite acidosis. This increase in acid body production must be viewed in the light of an expression of the toxemia and not as the cause of the condition. The clinical picture may, however, be greatly influenced by the production of these substances.

Acute Upper Respiratory Tract Infection Associated with Acidosis. For several years past we have been experiencing epidemics of acute upper respiratory infection associated with evident inflammation of the tonsils, pharynx, naso-pharynx and nasal mucous membranes resulting in an injection and glazed appearance of the mucous membranes. It is frequently unaccompanied by follicular involvement of the tonsils. Nausea and vomiting are early manifestations and may disappear in one

or two days or in fatal cases persist throughout the course of the disease. Usually by the second day after the beginning of vomiting an acetone odor is noted in the breath. Many of the cases early show acetone and a considerable number diacetic acid in the urine. Not infrequently, however, the mild cases run their course without these latter findings. Diarrhea is present in some of the cases and may be accompanied by marked evidence of intestinal fermentation. The majority of our cases, however, were associated with a flat abdomen and constipation. In the severe cases hyperpnea develops and may be the most marked of all of the symptoms. The children develop anhydremia, features are drawn, eyes sunken, skin becomes dry and loses its turgor, they become apathetic and later stuporous, and unless relieved develop profound coma. The epidemics were usually seen during the winter and spring months, and more commonly affect children between the ages of 1 and 6 years. Younger infants, however, may develop this condition and it is not infrequently seen in the breast-fed. Physical examination in the absence of tympany usually reveals more or less rigidity of the abdominal muscles and an enlarged liver. In some of the severe types icterus may develop. Throat cultures, taken early in the course of the disease, have in most instances in my cases shown a short chain small streptococcus.

What relationship the acute infection in the pharynx bears to the clinical picture is undecided. In all probability it is due to a general toxemia. However, the possibility of an acute gastritis, with secondary starvation, due to the inability to retain food, must not be overlooked as the possible cause of the acidosis.

A number of our cases were seen in children who had previously had a radical tonsil operation performed, so that they could not be related to pus infections of

the tonsils themselves. Rachford,[1] who has described these cases under the title of "Epidemic Acid Intoxication," has seen a large number of fatal cases. This has not been our experience in the Chicago epidemics, most of the cases running a comparatively short course of from two to six days, ending in a rapid convalescence.

Pulmonary and Cardiac Diseases. In the pneumonias, empyema and bronchial asthma and other types of massive involvement of the pulmonary tissues, and in cardiac diseases associated with decompensation, carbon dioxide frequently accumulates in the blood in amounts in excess of the normal, resulting in a carbon dioxide acidosis. This leads to increased stimulation of the respiratory center, with resulting hyperpnea. The carbon dioxide tension in the alveolar air is diminished but on account of the peculiar character of the acid causing the acidosis there is no diminution in the alkali reserve. In some cases Howland and Marriott found that lactic acid was produced as a result of the partial asphyxia and in these cases a slight diminution in the alkali reserve was noted.

Chronic Upper Respiratory Infections. Sedgwick[2] directs attention to the surprising results obtained following removal of the tonsils and adenoids in some of his cases of recurrent or periodical vomiting. He found that not infrequently tonsillitis or rhino-pharyngitis preceded the attacks. The possibility of focal infection should therefore be borne in mind in the recurrent types of vomiting associated with acidosis. The type of recurrent vomiting, probably a toxic neurosis which resembles migraine of later life in many of its clinical and laboratory findings and familial tendencies, should be given due consideration in an analysis of this group.

Acidosis Associated With Diarrheal Conditions. It has been shown by Howland and Marriott,[3] and the

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1 Rachford, B. K.: Arch. Ped. vol. 37, Nov., 1920, p. 651.
2 Sedgwick, J.: Jour. Dis. of Children, iii, 1912, 209.
8 Howland and Marriott: Penna. Med. Jour., April, 1918.

work has been confirmed by Yllpö[1] and Schloss,[2] that, in the diarrhea of infants accompanied by profuse watery stools (intoxication, cholera infantum, anhydremia, etc.), a severe acidosis may result. It is not present in all cases of diarrhea but may be a very serious and often a fatal complication. The carbon dioxide tension of alveolar air is low; frequently it sinks to 20 mm. or less, the alkali tolerance is much increased, the alkali reserve is greatly diminished and the hemoglobin dissociation curve is altered. Children suffering from this form of acidosis show, in addition to the other symptoms referable to diarrhea and anhydremia, hyperpnea and psychic changes.

The cause of this acidosis is not always evident, although it is in all probability frequently influenced by an insufficient intake of food, starvation due to vomiting, anhydremia through loss of fluids by vomiting and diarrhea, and excessive loss of bases through the intestines and disturbances in carbohydrate metabolism may all be factors. Although the acetone bodies are at times increased, they may be within normal limits. In a considerable number of cases the inorganic phosphorus of the serum is increased. The anhydremia results in a diminished blood volume, with a secondary diminished blood flow especially in the capillaries, which leads to a partial tissue asphyxia and increased lactic acid production.—Marriott.

From our present knowledge we are led to believe that increased production and an insufficient elimination of acids is, therefore, largely responsible for the development of acidosis in the course of the diarrheal conditions.

Acidosis Secondary to Starvation. It is a well-known fact that in partial starvation acetonuria is a frequent finding. The same is true when patients suffering from infections are underfed. The presence of acetone in

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[1] Yllpö: Ztschr. f. Kinderh., Vol. xiv, 1916.
[2] Schloss, O.: Amer. Jour. Dis. of Children, Vol. 13, 1917, p. 218.

these cases does not signify that they must necessarily be in a state of acidosis.

The work of Yllpö,[1] in 1914, was a stimulus to the study of this subject in new-borns. He claims to have found that the human organism at birth, is in a state of "physiologic acidosis;" as evidenced by chemical examination of the blood. Sehan[2] studied ten new-borns, from 1 hour to 8 days of age, and found that during the first few days, when the baby received practically no food at all, there was no definite decrease in CO_2 tension, which is indicative of acidosis such as Yllpö claims to have found. His results with the alkali tolerance tests in the normal new-borns led him to believe that they were not suffering from acidosis. He also found that acetone is practically absent from the urine of a normal new-born.

Veeder,[3] as a part of a general study of acetone body acidosis in childhood, made a number of experiments to obtain figures of the degree of acetonuria resulting from inanition. He believes that there is but little increased output of acetone bodies during a period of twenty-four hours inanition in a *healthy* child. In the cases in which inanition was terminated at the end of twenty-four hours, there was a continued increased elimination of acetone on the following day, which in some instances was greater than the amount eliminated on the day of the inanition. This fell rapidly, so that by the second day normal figures were again obtained.

In the remaining cases the inanition period was continued for a second twenty-four hours before the child was placed on the original diet. A rapid increase in acetone elimination took place during the second twenty-four hours.

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[1] Yllpö, A.: Neugeborenen-Hunger und Intoxikationsacidosis, J. Springer, Berlin, 1916.

[2] Sehan, M.: Am. Jour. Dis. of Children, Vol. 18, 1919, p. 42.

[3] Veeder, B.; Johnson, M. R.: Am. Jour. Dis. of Children, V. 13, 1917, p. 89.

Veeder does not believe that there is a relationship between the degree of acetonuria and the state of the child's nutrition, thin children giving the same results as fat ones. He found that the amount of beta-hydroxybutyric acid (in terms of acetone) eliminated was as a rule from two to four times as much as the quantity of combined acetone and diacetic acid.

The nitrogen and ammonia of the urine was determined daily. There was at most a very slight actual increase, or percentage increase, in the ammonia elimination on the first day of inanition, however, later a marked increase took place.

Gamble and his co-workers,[1] using as subjects epileptic children who were being fasted as a therapeutic measure, undertook to determine the extent to which the reduction of the plasma bicarbonate during fasting is due (1) to depletion of the inorganic base of the plasma, and (2) to the concentration of ketone acids developing in the plasma. It was found that the total inorganic base concentration was during fasting accurately sustained at its usual average value. The reduction of bicarbonate was thus apparently due entirely to the concentration of abnormal organic acids in the plasma. This inference was sustained by direct measurements of the concentration of ketone acids in the plasma. It was also found that small amounts of sucrose by mouth brought about a restoration of the bicarbonate concentration to its usual size. From these data it is inferred that glucose rather than bicarbonate is the logical agent in the treatment in non-diabetic ketosis.

Abt[2] has described a series of cases of acid intoxication recurring in infants previously healthy, at about the weaning period. In one family two children had previously died from this affection, the case described

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[1] Gamble, J. L., Ross, S. G., and Tisdall, F. F.: Transactions Amer. Ped. Society, 1922.

[2] Abt: Amer. J. Med. Sc., 147, 1914, 86.

being the third infant born to this family. W. F. Orr had reported a series of five children in the same family who died with symptoms similar to those described by Abt. The illness usually occurs in large, robust, previously healthy infants—in some cases the infants had shown a stationary weight curve before the onset. As in some of his cases I have seen three cases occurring in infants who suddenly weaned themselves because of dissatisfaction with the food from the breast, following which they also declined to take sufficient artificial food. As several of our cases have occurred before the close of the period of lactation the question arises as to whether the insufficient breast milk supply may not have led to partial inanition which became acute with spontaneous refusal of artificial food, resulting in complete starvation. The possibility of infection as a precipitating factor must, however, not be overlooked.

Post-Operative Acidosis. Morriss[1] found that the capacity of the plasma for combining with the carbon dioxide is decreased after ether and chloroform anesthesia; in other words, one influence of the anesthetic is toward depletion of the alkali reserve.

Jeans[2] found that the acetone bodies of the blood were somewhat increased after operation in about two-thirds of the children studied, the maximum amount being found in most instances about twenty-four hours after operation. The plasma carbonate was reduced in about two-thirds of the cases, the greatest reduction occurring in most instances about twenty-four hours after operation. When more closely compared there was found to be no close relation between the increase of acetone bodies and the reduction of plasma carbonate. In most instances, especially in those cases in which the plasma carbonate was much reduced, the acetone bodies were entirely inadequate to account for the degree of reduc-

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1 Morriss: Jour. A. M. A., May 12, 1917.
2 Jeans: Papers of the St. Louis Clinic, 1917.

tion of plasma carbonate. The undetermined acid factor was apparently of much greater importance than the acetone bodies in the reduction of reserve alkali. The starvation incident to operation seems to play no part in the production of this undetermined factor.

Acidosis of Renal Origin. The ability of the kidneys to excrete acid, especially acid phosphate, is one of the chief defensive mechanisms of the body. Failure of this mechanism even without increased production of acids, results in acidosis. Occasionally in nephritis acidosis occurs and Howland and Marriott[1] have shown that when acidosis is present there is an accumulation of unexcreted phosphate in the blood plasma. There is no evidence that any abnormal acid is produced. In these cases of acidosis where hyperpnea is present, the CO_2 tension in the alveolar air is lower than normal, the alkali reserve of the blood is depleted, and there is an increased "alkali tolerance." No acetone bodies are to be detected in the urine and the ammonia excretion is normal or diminished. Failure to produce ammonia to neutralize the retained acid is an additional factor in the production of the acidosis of nephritis, and represents a weakening of another of the body's defenses.

Acidosis Secondary to Burns. Acidosis frequently develops following severe burns. Marriott has found this to be a mixed type and is due in part to acetone bodies and in part to lactic acid.

Symptoms. The onset is usually relatively insidious. An early and the most important sign is *hyperpnea,* characterized by deep, pauseless respirations. The rate is usually moderately increased as compared to the great increase in depth. It is both thoracic and abdominal, the great amplitude of the respiratory excursion causing considerable effort and often the accessory respiratory muscles are brought into action. This type of breathing

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[1] Howland and Marriott: Arch. Int. Med., Vol. xviii, 1916, p. 708.

is described as "air hunger" and is the manifestation of an effort to increase pulmonary ventilation. Eventually, in fatal cases, there is exhaustion of the respiratory center, the respirations become feebler with occasional deep gasps and finally cease. Because of the increased aëration of the blood, the lips and cheeks are often a bright, cherry red.

The sensorium is markedly affected. At first, there is extreme restlessness and excitement, later apathy, drowsiness and stupor develops. Anhydremia frequently develops, manifested by sunken eyes, depressed fontanel and dry skin hanging in folds, more especially in the presence of repeated vomiting. The face is anxious and pinched. Diarrhea may accompany the acidosis. A "fruity" odor of the breath is sometimes present—this is characteristic of acetone body acidosis only. Dextrose alone, or in combination with lactose and galactose, may appear in the urine,[1] in the particular form of acidosis occurring in anhydremia. The degree of leucocytosis and fever depends upon the accompanying disease. Hyperpnea without cyanosis is the only reliable and pathognomonic clinical symptom. The various laboratory tests show an impending acidosis before any symptoms are manifest. *The laboratory tests* confirm the diagnosis. They give positive evidence of the presence or absence of acidosis, even in absence of characteristic symptoms.

The determination of the carbon dioxide tension in the alveolar air is a relatively simple, bedside procedure, if the colorimetric method of Marriott[2] is used. In infancy the normal carbon dioxide alveolar air tension varies from 37 to 45 mm. Tensions between 30 and 35 mm. indicate a mild degree of acidosis, below 30 mm.

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[1] Schloss, O.: Amer. Jour. Dis. of Children, xiii, 218, 1917.
[2] Marriott: Jour. A. M. A., lxvi. 1594, May 20, 1916. Howland and Marriott: Am. Jour. Dis. of Children, xi, 309, 1916.

a moderate, and below 25, a severe acidosis, and below 20 mm. an extreme acidosis.[1]

More difficult, but more accurate are the determinations of the bicarbonate content of the plasma by the Van Slyke method.[2] The amount of CO_2 given off is measured after strong acids are added to a certain volume of blood to liberate the CO_2. In infants the plasma contains 50 to 70 volumes per cent. of combined CO_2. Adults have about 10 per cent. more. In infants amounts less than 45 indicate acidosis. The results, if multiplied by 0.7, approximate alveolar carbon dioxide tension in ·millimeters determined according to the method of Marriott. As mentioned previously, the excessive H ions stimulate respiration and the excessive acids in the blood decompose the carbonates so that CO_2 is blown off through the lungs, lowering the amount combined in the blood. Consequently, this test indirectly measures the available alkali reserve of the blood.

Sellard's[3] test is to determine the alkali tolerance of the individual. Under normal circumstances, the ingestion of small quantities of sodium bicarbonate by mouth increases the amount of this substance in the blood and the excess is rapidly excreted in the urine, causing the reaction of the urine to become amphoteric or alkaline to litmus temporarily. Marriott recommends the use of brom-cresol purple in 2 per cent. alcohol solution; a drop or two of this solution is added to a small amount of urine in a test tube. This indicator changes from yellow to purple at about the normal reaction of the urine (PH_6). When the urine turns this indicator purple, it indicates that acidosis is not present, or that the acidosis has been corrected. If the indicator does not turn purple, it may or may not mean the presence of acidosis. This is a simple test which can easily be com-

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[1] Howland and Marriott: Bull. Johns Hopkins Hosp., xxvii, 63, 1916.

[2] Van Slyke, D. D.: Jour. Biol. Chem. xxx, p. 347, June, 1917.

[3] Sellards: Johns Hopkins Hosp. Bull. xxv, 101, 1914.

pleted in the office. An alkaline reaction of the urine can be brought about in normal infants by administering 2 to 3 Gm. of bicarbonate, and in older children and adults by 5 Gm., usually within an hour after administration, this will change the indicator purple. In acidotic states four to ten times as much is necessary, because first the bicarbonate given must replenish the depleted alkali of the blood and tissues, before being excreted in the urine.

Another method devised by Sellards[1] consists essentially in precipitation of the proteins from a small quantity of blood and adding a drop of phenolphthalein indicator to the evaporated filtrate. In acidosis, instead of a deep red color, a pink or no color is present.

Tests for acetonuria only apply to certain special varieties of acidosis. (Acetone-body acidosis.) The presence of such bodies are, however, not sufficient evidence when taken alone for diagnosis of acidosis; the reverse, however, holds true in that their absence does not exclude its presence.

Again, in special types of acidosis (increased production of organic acids or ingestion of strong mineral acids), there is an increased excretion of ammonia combined with these acids. Consequently, a quantitative estimation of the ammonia co-efficient in the twenty-four hour urine is a valuable test for acidosis. It should, however, be remembered that in the presence of nephritis the ammonia excretion may be normal.[2]

The first three methods described are those most practical for routine clinical investigation.

Prognosis. The prognosis varies with the precipitating etiological factors, the severity of the acidosis, and upon the underlying constitutional conditions. The result obtained is dependent first upon our success in the removal of exciting factors; second the overcoming

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[1] Sellards: *Ibid.*

[2] Henderson, Y.: Arch. Internal Med., xvi, 109, 1915.

of the intoxication; and third, so far as possible the relieving, or when possible, removal of the underlying factors. The early relief of starvation, by the institution of the proper diet, counteraction of infection, and the neutralization of the acetone bodies, is all that is usually necessary in the mild types secondary to starvation and infection. In children suffering from recurrent vomiting, sources of focal infection should be removed. In the acute types associated with diarrhea the prognosis is grave. Diabetes and chronic nephritis must always be reckoned with as extremely serious factors. In cases of acute nephritis of moderate severity the prognosis is better.

Treatment. The treatment of acidosis must first be directed to the prevention of a further production of more acids; second, to replenishing the alkali reserve; third, to the elimination of the acids and their salts; and fourth, to the treatment of the underlying factors, whether they be acute infections, starvation or organic diseases.

Water Administration. Large quantities of water should be administered by mouth, if it is tolerated. When not retained, subcutaneous or intraperitoneal administration of normal saline or Ringer's solution is to be recommended, the latter as often as every six hours, if necessary, during the early treatment. The total quantity of all fluids given should be recorded daily and every effort made to administer maximum quantities.

In the presence of *acetone body acidosis* glucose should be given in all cases except those due to diabetes. Three methods of administration are available: by mouth, rectum, or intravenously. By *mouth,* a 5 to 10 per cent. solution may be administered every two to four hours, in quantities depending upon the age of the child and the tolerance of the gastro-intestinal tract. By *rectum,* a 5 per cent. solution is best employed, which may be administered intermittently at intervals of three or four

hours, quantities varying from 60 to 120 mils (two to four ounces) being used, or in young infants and older children who are not too restless the drip method may be used. When there is a tendency to return the glucose solution it may be necessary to compress the buttocks for at least one-half hour after each administration. A careful record should be kept of all solutions administered by this method and so far as possible the retained fluid estimated. In most cases a liter can be administered in the course of twenty-four hours. For *intravenous* use, 5 or 10 per cent. of glucose in Ringer's or normal saline solution may be used, depending upon the size of the child, from 60 mils in small infants, to 300 mils in older children, may be injected at one time, to be repeated as indicated.

Intraperitoneal Injection. For this purpose equal parts of 4 per cent. glucose solution and Ringer's solution may be used, making a 2 per cent. glucose and 0.40 per cent, saline solution. Stronger solutions are hypertonic. Fifty to 500 mils may be administered by the gravity method.

Alkali Therapy. In our experience we have not favored the use of alkalies in the types of acidosis due to concentration of ketone acids and this agrees with the more recent experiences of Marriott,[1] Gamble,[2] and Schloss.[3] Glucose rather than bicarbonate is the logical agent in the treatment of non-diabetic ketosis. The same is true of the lactic acid type. Marriott found that when sodium bicarbonate is given, the amounts of sodium and sodium ions in the blood are increased and this increase disturbs the balance between the sodium, calcium and magnesium ions. If administered sodium bicarbonate should be given by mouth or intravenously. By

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[1] Marriott, W. McKim : Personal communication.

[2] Gamble, J. L., Ross, S. G., and Tisdall, F. F. : Transactions Amer. Ped. Society, 1922.

[3] Schloss, O. M.: Trans. Amer. Ped. Society, 1922.

mouth from 1 to 2 grams may be given at four-hour intervals, dissolved in water. For intravenous use a 2 per cent. solution is best employed as it is isotonic with the blood. · The same amounts of solution can be administered intravenously as recommended for the administration of glucose.

Howland and Marriott found that the simplest indication that sufficient alkali has been administered is evidenced by shifting of the reaction of the urine to normal. When it becomes amphoteric or alkaline to litmus, we may consider that a slight excess has been given. Further administration should then be withheld unless the urine again becomes more acid than normal. When the carbon dioxide tension of the alveolar air is above 30, recovery is usually spontaneous and soda is unnecessary. Edema, tetany and even convulsions have been noted following the administration of large doses of soda, more especially in young infants. These usually disappear with the cessation of the soda medication.[1]

Diet. While the administration of glucose as recommended may carry the patient over the first emergency period, insufficient amounts of carbohydrate are given in this way to meet its needs. Efforts should therefore be directed toward the further administration of carbohydrates by mouth as soon as the condition of the stomach permits. After a short period of rest, the time of which will be indicated by the ability of the stomach to retain inert fluids, which in most instances is not over six to twelve hours, further administration of sugars and cereals should be begun. Thick cereal pastes, to which cane sugar or maltose dextrose compounds have been added, are usually best retained. They may be given in small quantities at two-hour intervals, or at

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[1] The treatment for convulsions due to an excess of sodium ion in the body consists in pushing water and the administration of magnesium sulphate intramuscularly or subcutaneously. (See Spasmophilia.)

longer intervals with the administration of corn ór maple syrup, or honey in teaspoonful or larger amounts. Malted milk is often well taken. Milk chocolate and plain candy, such as caramels, are valuable and are usually well taken by children who have eaten them previously and have a liking for them. Various plain wafers sold on the market may be tried on the second day. Fruit juices and sirups, more especially orange juice, should be given in small quantities. If orange juice is retained, larger quantities, diluted with sweetened water, should be given and continued. Skim milk or, better, skim buttermilk can usually be given by the third day. Whole milk and fats should be withheld until the child is convalescent.

Medical Treatment. About the only conditions requiring further medication are gastric and intestinal distention and the retention of decomposing food in the intestinal tract, vomiting and syncope.

To clear the intestinal tract milk of magnesia or some other mild saline laxative, together with enemata, are best. For vomiting in the presence of gastric distention lavage with a weak bicarbonate solution is often most beneficial. Small doses of carbolic acid ($\frac{1}{8}$ to $\frac{1}{4}$ minim.), administered at two-hour intervals for three or four doses, together with tincture opii camphorata for the relief of vomiting and restlessness, are indicated. In the presence of syncope it may be necessary to administer cardiac and respiratory stimulants.

PART IX.

Anemias of Infancy.

A PROPER knowledge of the normal blood picture in early life is necessary in order to recognize pathological changes in the anemias.

In the blood of the *new-born* we find the following:

The hemoglobin ranges from 110 to 140 per cent. as a rule. The erythrocytes are increased to from five to seven million, or over.

These findings begin to decrease usually by the fourth day and toward the end of the first month the hemoglobin content and erythrocytes have arrived at the usual level of infancy. Nucleated reds may be present up to the third to sixth day. The leucocytes are often increased up to 30,000, averaging 70 per cent. polymorphonuclears and about 20 per cent. lymphocytes. Within a few days the proportions quoted below as averages for infancy are more nearly approximated. Myelocytes may be present for a few weeks.

In the *infant's blood* we find the following:

A slight decrease of erythrocytes and hemoglobin below the adult level, may be present. The red cells range from 4.5 to 5.5 million; the hemoglobin from 70 to 95 per cent.

The leucocytes average about 12,000 or 13,000. There is a preponderance of lymphocytes (60 per cent. as compared with 30 per cent. at the 15th year), and a small number of polymorphonuclears (30 per cent. as compared with 70 per cent. at the 15th year). The transitionals average between 8 and 10 per cent. The reversal of these percentages occurs by the 6th year.

(405)

Definition. Anemia is characterized by a decrease of the erythrocytes or hemoglobin, or both, in the blood. The hemoglobin has the most important function of the blood elements, namely, carrying oxygen, and so its determination affords the most important method for ascertaining the functional capacity of the blood. Not infrequently infants have a pale appearance and are seemingly anemic, but an examination of their blood reveals a normal red count and hemoglobin content; this phenomenon may be ascribed to an angiospastic pallor of the skin.

Etiology. Anemia may develop from any of the factors producing it in the adult, but it tends to follow in the wake of less severe causes and there is a tendency to more rapid recovery in the absence of constitutional anomalies. Probably the most frequent causes of anemia in infants are nutritional disturbances, directly or indirectly due to faulty dietary and iron metabolism. Rickets goes hand in hand with this group of anemias. Especial stress will be placed upon this group in the discussion because of its relevant bearing upon nutritional disorders in infancy. Other common causes of infantile anemia are infections, both acute and chronic, and hemorrhagic conditions.

A convenient working etiological classification of the causes of anemia in infancy follows:

1. *Congenital conditions* resulting in defective blood formation.

> Diseases of the mother during pregnancy, due to improper diet, eclampsia, syphilis, tuberculosis, diabetes, etc.

> Prematurity and other conditions with resulting hypofunction of the hematopoietic system. Developmental defects in the blood-making organs and glands of internal secretion (aplastic anemia, cretinism).

2. *Primary Hemorrhage.*

3. *Acquired.*

 A. Conditions resulting in diminished blood formation:

  Due to improper hygiene, with resulting disturbances of nutrition;

  Due to dietetic errors (unduly prolonged lactation), (athrepsia, rickets and scurvy);

  Diseases of the hematopoietic system (pernicious anemia, splenic anemias, leukemia).

 B. Increased blood destruction, due to toxic causes:

  Bacterial toxines (acute and chronic infections);

  Parasitic toxines;

  Endogenous toxines (nephritis, hemolytic jaundice);

  Chemical poisons (arsphenamin, phosphorus, mercury);

  Roentgen rays.

 C. Secondary hemorrhages, due to degenerative blood-vessel changes as a result of the causes enumerated in Group 3.

Diseases of the mother and fetus, tending to premature birth, have a direct influence on the postpartum blood findings. The blood pictures, however, vary considerably, depending upon the etiological factors involved. There usually is a predominance of the regenerating blood cells which may be of the embryonal or post-embryonal type.

The characteristic type of cells produced by the blood-forming tissues in the fetus are as follows, normally macrocytes, myelocytes and megaloblasts, and are produced by the fetal blood-forming tissues. Incomplete types of cells, such as the microcytes, poikilocytes, polychromatophiles, and normoblasts, may be found in both the embryonal and post-embryonal stages. In the infant

whose immature tissues have only recently been of the fetal type, a reversion to embryonal blood cells is a common finding in the anemias of even moderate severity, and is of much less significance than of the same findings in adults.

Anemia in the mother during pregnancy is a cause of anemia in the fetus at birth. This condition in the mother may result from improper diet, a severe acute disease suffered during pregnancy, or from constitutional diseases of the mother, as syphilis, tuberculosis, nephritis, etc.

Striking is an anemia which develops quite regularly in *prematures* during the first three months of life. In contrast to full term infants, in the prematures there are a greater number of nucleated red blood corpuscles, a more frequent appearance of myelocytes during the first days of life, and a lesser absolute and relative leucocytosis. There is also a distinct and very early hemoglobin impoverishment of the blood, which reaches its maximum in about the third to the fourth month. In the premature and twins it may fall rapidly to 50 per cent. or lower. This anemia is usually of the chlorotic type, the color index being usually 0.4 to 0.6. These infants develop a severe secondary anemia following infections, etc., much more readily than full term infants, even in the later months of the first year. Kunckel[1] believed that the chlorotic-like anemia of prematures was due to a disturbance in the hemoglobin metabolism as well as a deficient iron storage. Lichtenstein[2] believed it due to a hypoplastic condition resulting from insufficiency of the hematopoietic organ. Hugounenq[3] has shown that the greater proportion of the iron is deposited in the fetus during the last three months of pregnancy. The premature therefore fails to get the required

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[1] Kunckel: Zeitschr. f. Kinderh., vol. 13, 1915, 101.
[2] Lichenstein: Svenska, La Karesa As Kapets Handlinger, 43, No. 4, 1917.
[3] Hugounenq: Jahrb. f. Kinderh., 51, 121.

amount of iron deposited in its liver (iron depot). Twins must divide the iron which the mother can furnish and so also have a congenital deficiency.

Congenital hypoplasia and imperfect development of the bone marrow, which prevents the hematopoietic tissues from properly functioning and producing sufficient vascular elements, may cause a distinct condition (aplastic anemia) or merely be a contributing cause in the development of various anemias in infants. Probably the reason some children develop severe forms of anemia and others only a slight degree under the same conditions, lies in a constitutional weakness of the blood producing organs of the former. Instances have occurred in which several children in the same family have possessed hematopoietic organs which were not capable of functioning sufficiently, death or severe anemias resulting. It is reasonable to assume that there may be congenitally deficient bone marrow just as there is congenitally deficient thyroid.

In infantilism and congenital anomalies of the glands of internal secretion (cretinism) anemias are usually marked. In the former deficient development of bone and bone marrow is cause enough for the anemia accompanying these conditions.

Anemias following *hemorrhage* are of the secondary type with a tendency to reversion to the embryonal blood picture. The most common causes of early blood loss in infants are bleeding from the cord and intracranial vessels. Somewhat later melena, hemophilia and sepsis may become evident causes, and in the later months purpura and scurvy must be considered.

Faulty hygienic surroundings may alone cause considerable anemia; however, there are usually other associated factors.

Lack of "mothering" and institutional overcrowding predispose to secondary infections.

As mentioned previously, *nutritional disturbances* are the cause of the largest percentage of anemias in early life. There is nothing specific about the degree of anemia which a nutritional disturbance will cause. In some cases a severe degree of anemia will develop with a severe nutritional disturbance, in others the anemia will be slight and vice versa. From about the seventh month to the end of the second year (at the end of the nursing period) there are more anemias with relatively grave blood pictures than at any other age. It appears, therefore, that there is a predisposition toward a functional insufficiency of the hematopoietic system at this age which predisposes to anemias. This is somewhat analogous to the diseases of the skeletal and nervous system of this period, namely, rickets and spasmophilia. Improper artificial nutrition, metabolic and gastro-intestinal disturbances and poor hygienic surroundings probably play an important rôle in the etiology. Premature infants, twins and those who have not completely recovered from previous hemorrhages are especially predisposed.

Faulty feeding may produce anemia in several ways.

First, there may be an insufficient amount of iron in the food. The new-born has an iron reserve or storage in his tissues of which the liver is the most important depot, which is highest at birth and then gradually falls until a minimum is reached at the end of the lactation period. This iron reserve prevents an iron deficiency while the infant is being fed on iron-poor milk. However, if the iron-deficient food is fed over too prolonged a time, without the addition of mineral rich food to the diet, an anemia is prone to develop. Naturally, in some infants this condition develops sooner than in others, depending upon the original amount of stored iron surplus and the demand upon this reserve.

Second, faulty feeding leading to chronic functional intestinal and metabolic disturbances may cause anemia. This may be due to lack of absorption of enough food.

In most conditions of poor or impaired nutrition all the organs of the body suffer to some extent. Thus the bone marrow may function deficiently under these circumstances and produce fewer erythrocytes, or erythrocytes deficient in hemoglobin.

Improper feeding may result in certain severe nutritional disorders, as rickets and scurvy. In these conditions the more or less severe anemia, which always accompanies them, may be due to the same etiologic factors (poorly balanced diet, improper hygiene, etc.) or result from the diseases themselves or both. Thus the hemorrhages in scurvy may lead to anemia and the pathology in the bone marrow of rickets interfere with its hematopoietic function.

Of the causes of the primary anemias little positive information is at hand. They are, in all probability, toxic in nature with a secondary effect on the blood-making organs, and may be exogenous or endogenous in origin.

The *bacterial toxines* are second only in importance to the nutritional disturbances as etiological factors in the production of secondary anemias. Among the most important of the acute infections are diphtheria, scarlet fever, tonsillitis, pyelitis and otitis, and septic infections. Among the chronic, tuberculosis and syphilis play the most important rôle in infancy. *Parasitic* and *chemical toxines* are of far less importance in infancy than in older children, except those following arsenical poisoning.

Among the *endogenous toxines,* those resulting from nephritis are frequent causes of secondary anemias.

Iron Metabolism. The iron content of both human and cow's milk is small and not sufficient to meet the requirements of the developing infant. However, there is an iron depot in the liver[1] in which is deposited enough iron by the time of birth to last until the infant can digest foods containing iron. If this original supply is abnormally small (as in prematures, etc.) or there is

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[1] Burge: Zeitschr. f. Physiol. Chemic., xiii, 399, 1889.

an unusual drain upon it (as in hemorrhages, infections, etc.), the supply may be used up before iron-containing foods are added to the diet.

Human milk contains 0.00015 per cent. of iron and cow's milk about 0.0007 per cent. Iron is absorbed by the small intestine and excreted by the large intestine. Inorganic, as well as organic iron is easily absorbed. But the fact that iron given *per os* is absorbed into the circulation and excreted into the large bowel is not proof that iron is really used by the organism. However, the administration of iron medication *per os* has a markedly beneficial effect on certain types of anemia.

In 100 Gm. of ash in the newborn there are about 0.8 Gm. of Fe_2O_3; 38 to 40 per cent. of this iron is in the blood; the remainder is deposited in the tissues (Hugounenq).[1] The actual amount of hemoglobin in the body rises from birth, but the amount as compared with the body weight diminishes. The amount of iron deposited in the tissues, especially in the liver of the newborn, diminishes soon after birth. In other words, the iron in combination with hemoglobin, or the hemochromogen radical, increases from birth, but the iron not so combined (reserve iron) diminishes. It is thus easy to understand why the new-born should have such a large amount of hemoglobin and reserve iron, and that this hemoglobin iron should increase, for the relatively great body surface in the new-born requires a large amount of oxygen-carrying material. Therefore a large amount of hemoglobin must be produced. The hemoglobin iron is increased at the expense of the non-hemoglobin iron.[2]

Symptoms. There are no special subjective symptoms characterizing anemia in infancy. Many of the symptoms present can be attributed to the condition causing the anemia. The majority of cases may be grouped

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[1] Hugounenq: Jahrb. f. Kinderh., 51, 121.
[2] Schwarz, H. and Rosenthal, N.: Arch. Ped., 37, 1, 1920.

into two more or less defined types. The thin underweight infants, who seem never to have thrived from the time of their birth—they are especially liable to nutritional disturbances and infections. The second group may be but little under weight and present a well rounded appearance; however, on closer examination they show a more or less marked pallor, and in the extreme cases of the fat type, their skin is of a greyish or waxy yellow tint and at times seemingly semitransparent. The mucous membrane findings are of varying intensity and are of the same degree as those of the skin. The majority of cases belong to the second group and include many of the premature and rachitic infants and those with low grade infections. Edema occasionally appears about the face, trunk and extremities in the severe forms. The onset may be sudden, or gradual, depending upon the etiologic condition, i.e., sudden, following infections and hemorrhages; gradual, following nutritional disturbances, or again, as in prematures, it may be present from birth. Anorexia, listlessness and muscular weakness may be present. Occasionally the appetite may be capricious. The state of nutrition also varies with the cause. The infant may be considerably underweight although at first appearance this may not be evident. There may be gastro-intestinal disturbances. Hemic murmurs are not as frequent as in older children and adults. In the severe anemias, petechiæ may occur in the skin and mucous membrane, but severe hemorrhages are unusual except possibly epistaxis. More severe hemorrhages may, however, occur following ulceration of other mucous membranes.

Splenic enlargement associated with anemia is a very frequent finding during the first year of life and its presence is of great diagnostic value. Not infrequently there is an increased consistency with only moderate enlargement and again in the presence of sepsis and other infections there may be a considerable enlargement but

the spleen may be so soft that it is difficult to palpate. The spleen is usually considerably enlarged in the severe secondary anemias from all causes. It may decrease in size before the blood itself has reached normal levels. A large hard spleen in the first three months of life should lead to an examination for the presence of syphilis. Other common causes of splenic enlargement during the first six months are septic infection, tuberculosis and various types of subacute infections. The anemias due to various constitutional diseases, such as rickets and status thymico-lymphaticus, are usually associated with splenic enlargement. The most marked enlargements accompanied by anemia occur in von Jaksch's anemia, the leukemias, Banti's and Gaucher's diseases. Hemolytic jaundice is also accompanied by enlargement of the spleen.

The *enlargement of the liver,* although not infrequently present, is rarely a predominating feature in the clinical picture.

Hyperplasia of the lymph glands is directly dependent upon the underlying cause of the anemia.

Blood Findings. The changes in the blood picture are the most important factors in the differential diagnosis of the anemias of infancy.

In the *simplest secondary anemias* resulting from hemorrhage, infections (toxins) or nutritional disturbances, there is usually a reduction of both hemoglobin and erythrocytes to about the same degree. In the more severe forms, anisocytosis, poikilocytosis, polychromatophilia and normoblasts are not unusual. Usually very few embryonal (fetal) blood elements are found, such as megaloblasts, macrocytes, and myelocytes. The leucocyte count varies, depending on the causative factor.

Following hemorrhage a relative leucocytosis is usually noted. In the acute infections they vary according to the type of infection and the ability of the organism to react. Individual infants vary greatly in the response

of their hematopoietic systems to the same type of infections; in the acute septic infections usually an absolute increase in the neutrophiles is seen, while in tuberculosis, syphilis and influenza a low white count and at times a leukopenia with an absolute increase of lymphocytes may be noted. Subacute and chronic focal infections often show little or moderate increase in the total count with a predominance of lymphocytes.

The so-called *chlorotic type* of anemia—the common type seen in prematures, twins, and the poorly fed in the early months of life—probably has a similar foundation in many instances and presents the following blood changes: The hemoglobin is greatly diminished but the red count rarely falls below 3,000,000 and is more frequently between 4,000,000 and 5,000,000. The color index is usually between 0.4 and 0.6. The leucocyte count approximates the normal, ranging from 6000 to 10,000. There is a varying tendency to lymphocytosis at times. However, this should not be misinterpreted because of the normal high mononuclear count in the young. The red cells only occasionally show poikilocytosis and anisocytosis. Nucleated reds are rarely found. Blood platelets range between 200,000 and 300,000, the blood volume tends to remain normal and the spleen is usually not large.[1]

In the type described as *von Jaksch's anemia,* a condition occurring in infants and children, a marked anemia, moderate enlargement of the liver, and great enlargement of the spleen and sometimes hypertrophy of the superficial lymph nodes are present. The blood picture is characterized by a great diminution in the red cells and the hemoglobin, and a persisting leucocytosis of varying degrees. Nucleated red cells are nearly always present, sometimes in large numbers, and occasionally myelocytes are seen. The hemoglobin is usually below 50 per cent.

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[1] Schwarz and Rosenthal: *Ibid.*

and may be as low as 15 to 20 per cent. The red cells are generally 2,000,000 to 3,000,000 per c.mm., but may fall to 1,000,000, or lower. There is a high grade aniso-cytosis, poikilocytosis and polychromatophilia. The leu-cocytosis usually ranges between 15,000 and 50,000. The small lymphocytes are usually greatly increased more especially in the cases secondary to nutritional disturb-ances. Occasionally this picture is reversed in the pres-ence of infection. The condition is most frequently associated with rickets, but whether cause or effect or concomitant is not known. It is most common during the sixth to the fifteenth month, the age at which rickets is most frequently seen in the active stages. It is rare in the breast-fed and well-nourished infants. Syphilis and possibly tuberculosis are believed to play a rôle in some of the cases. Upper respiratory infections, chronic pyelitis, endocarditis, and like infections may cause a similar picture.

Most of the patients show a definite tendency to re-covery. The prognosis must necessarily be dependent upon the underlying cause and the presence of secondary complications.. In those who live, the abnormal blood picture may persist for a long period after the patient is clinically well. Grätz[1] regards it as an infantile form of Banti's complex. His view has received but little support. There has also been a tendency to call this condition "anemia splenica infantum." This is, however, undesirable, as in its present use "splenic anemia" is a descriptive term which is applied to a large number of unrelated conditions. For practical purposes and until the condition is better understood, it is preferable to speak of it as "von Jaksch's anemia," than "anemia pseudoleukemica infantum," the name originally given

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[1] Grätz: Unter dem Bilde der Anemia splenica verlaufende ex-tra-medullare Bildung von Blutzellen bei einem 3 jahrige Kind., Zentralb. f. allg. Pathol. u. pathol. Anat., 1909, xx. Cit. by Stettner.

to the condition, since the term pseudoleukemia has since acquired a more specific usage and the condition to which it refers is in no way related in so far as is known to this type.

Pernicious Anemia. This form of anemia is very rare in infancy. Well authenticated cases have, however, been reported. The anemia is of the hemolytic type. The red blood cells are greatly decreased in number and the hemoglobin, being set free, results in a marked decrease in its content. There is, however, a high color index in which this blood picture is different from that seen in any of the other types of anemia. There are normoblasts present and a high percentage of megaloblasts. The red cells are altered in other respects, showing poikilocytes, microcytes and macrocytes. Leukopenia is present in the majority of cases and a relative lymphocytosis of above that to be expected in infancy is usually present. In extremely leukopenic blood a noteworthy finding is the abnormally high percentage of large mononuclear non-granular cells. Myelocytes are almost always present. The spleen and lymph glands are usually not much enlarged.

Aplastic or hypoplastic anemia is dependent upon a congenital or acquired hypoplasia of the bone marrow. It is a rare condition and in its chief characteristics resembles pernicious anemia and is in all probability a form of this disease. The blood picture shows little effort on the part of the organism to reproduce the blood cells. It is characterized by a marked diminution in the hemoglobin and red cells. Regenerative forms of the blood cells are usually absent and a high grade leukopenia is usually present. The spleen more commonly shows moderate enlargement.

Among the more exceptional types of anemia seen in the young and which are always associated with splenomegaly are: *Gaucher's disease* or *primary splenomegaly*, in which the greatly enlarged spleen shows masses of

characteristic large vesicular cells with small eccentric nuclei, which block the sinuses of the spleen. Similar groups of cells are found in the lymph nodes, liver and bone marrow. It is characterized clinically by chronic splenomegaly, enlargement of the liver and a peculiar brownish or grayish discoloration of the skin. The anemia is usually of the chlorotic type and is more often of moderate degree. A diminution in the number of white cells is characteristic. It is probably congenital in origin, although usually not recognized until late in infancy or childhood.

Hemolytic icterus. Two types have been described: The familial or Minkowski type, which is often seen in several members of the same family. The main symptoms are an enlargement of the spleen and to a lesser degree of the liver. The most outstanding symptom is an acholuric jaundice. Increased fragility of the red blood cells is often marked and this condition must be classed as a true hemolytic icterus.

The second type, which is known as the acquired or Hayem type, may come on at any age and is usually associated with a considerable anemia and a decided enlargement of the spleen and liver. Fragility of the red cells and acholuric jaundice are constant findings. These conditions usually last throughout life and often show little effect upon the general health. No specific lesion has been demonstrated at autopsy.

Banti's disease or complex is discussed to complete the group but does not concern us in infancy, as it is a disease of later childhood and adults. Banti's complex and *splenic anemia*, as commonly described in the literature, are probably one and the same thing. Moschcowitz[1] believes that it is due to a fibro-genetic toxin, probably of intestinal origin, which attacks the organs draining the portal area, causing primarily a fibrosis of the spleen;

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[1] Moschcowitz, Eli: Jour. Amer. Med. Assn., lxix, 1045, 1917.

and if the toxin is sufficiently intense or the patient lives a sufficiently long time, it causes a cirrhosis of the liver. The sclerotic vascular changes in the mesenteric vessels are explainable on the same grounds. Finally, there develops an ascites due to atrophic changes in the liver. The course of the disease is slow, often covering a period of many years, with a gradually increasing weakness and pallor and digestive disturbances. A tendency to hemorrhages with a moderate anemia of the chlorotic type is usually present. The resistance of the red cells is unchanged and signs of a regenerating bone marrow, as evidenced by nucleated and reticulated cells, are slight or absent.

Leukemias while rare in infancy are of sufficient frequency to deserve mention. The types most likely to be seen are the acute forms.

Acute lymphatic leukemia is the most common form in early life. The symptoms are usually so severe and the course so rapid as to suggest an acute infection. In most cases there is a history of a preceding infection, such as tonsillitis, sinusitis, alveolar abscess, acute pulmonary infections, multiple abscesses, osteomyelitis, etc. Other cases develop in the presence of existing simple or secondary anemias, in the course of nutritional disturbances, rickets, scurvy, etc. Congenital syphilis has been described as a predisposing factor. Leukemia may, however, occur as a primary disease in infants previously healthy.

The onset may be abrupt with several general symptoms, as fever and prostration, or it may be more gradual, resembling a low grade infection. There soon develops a generalized glandular swelling, often first noticed in the cervical region, probably because the glands are more visible or due to the fact that they may be first involved when the source of the infection is in the region of the upper respiratory tract. However, the axillary, epitrochlear, inguinal and femoral glands usually soon

become involved, and roentgenographic studies will show involvement of the tracheal and bronchial glands and not infrequently the thymus. The glands vary greatly in size, from that of a pea to a walnut. They show little tendency to become tender and rarely suppurate. The spleen soon becomes enlarged, more often moderately, rarely reaching the size seen in the chronic forms. The liver is also enlarged in most of the cases. Both of these organs, as well as the other parenchymatous organs, show a marked infiltration with lymphoid tissue, either diffuse or in patches. The changes in the bone marrow vary, being slight in some instances. The gums become swollen and the findings in the mouth may resemble those of a severe scurvy. Bleeding occurs and not infrequently sloughing of the gums, tonsils and palate. Subcutaneous hemorrhages, petechial or larger ecchymotic areas, together with bleeding from the mucous membranes of the nose, stomach, intestines and bladder, usually occur during the course of the disease.

The blood picture varies in the different stages. In many instances great changes are noticed from day to day. At times there will be noted a considerable increase in white cells and this may change within a few hours to a leukopenia. The lymphocytes dominate the blood picture, often reaching from 90 to as high as 100 per cent. of the white cells, with a corresponding reduction in the other forms. Most cases show a very high percentage of the large type of lymphocytes. These cells, however, are frequently degenerate and offer great difficulty in staining for differentiation. The total white count usually runs from 25,000 to 100,000 cells in infants. At times, and more especially toward the end of the disease, they may almost disappear. The hemoglobin may be reduced to 10 to 30 per cent. and the red cells to 1,000,000 to 2,000,000. The diminished coagulability of the blood accounts in part for the great susceptibility to hemorrhage. The disease is usually ac-

companied by an irregular temperature curve and evidence of myocardial involvement.

The course usually covers a period of from two to four weeks from the time of its recognition, although some cases may run a longer course.

Splenomyelogenous leukemia, while more rapid in its course in the young is less acute than the lymphatic type and is an exceptional disease in infancy. Anemia and splenic enlargement are usually the first signs to be noted. There is more or less evident involvement of the liver. The lymphatic glands rarely attain a large size and may not be visibly involved. Asthenia is marked and an early manifestation, and is associated with a rapid, weak pulse, dyspnea and disturbances of the digestive tract. Hemorrhages into the skin and mucous membranes may be present at any stage of the disease. The total white count is greatly increased, at times varying between 50,000 and 500,000. The polymorphonuclear neutrophils are greatly increased, although at certain stages they may be in greater part replaced by myelocytes. Both the mono- and polymorphonuclear types of eosinophils are increased. The lymphocytes are increased, the small cells oftener predominating. Increase in both the mono- and polymorphonuclear basophils is one of the most characteristic findings of the disease.

The course of all forms of true leukemias of infants is toward a fatal termination. Most of the cases are complicated by secondary infections.

Prognosis. In the anemias of infancy the prognosis is dependent upon the nature of the cause and the ability of the individual infant to react. In the milder types associated with improper diet, poor hygiene and rickets, with removal of the cause, improvement is usually rapid. In those following acute hemorrhage of moderate degree, regeneration is usually rapid, unless there be some underlying constitutional condition, such as scurvy and purpura, when the prognosis must be guarded. In the

acute and chronic infections, more especially diphtheria, tuberculosis and syphilis, it will vary with the severity of the infection, the individual resistance and the institution of proper treatment. The same may be said of the cases following genito-urinary infections, such as pyelitis and nephritis. In the severe types of anemia associated with embryonal blood pictures, splenic enlargement, and grave constitutional involvement, the prognosis is always grave. In all types of anemia there is a tendency toward secondary infection due to lessened immunity.

Treatment. The treatment must be directed toward removing or curing if possible the exciting etiological factors, as well as toward remedying the existing anemias.

Prophylactically, much can be accomplished toward preventing certain of the anemias of infancy. · A complete clinical study of the patient must be made, including a thorough search for possible focal infections. The mouth, nose and throat, sinuses, respiratory, digestive and urogenital tracts must be carefully investigated. If any focal infection is found it must be thoroughly eradicated. The important part which alveolar abscesses may play in the secondary anemias, even in infancy, must not be overlooked.

The anemias of prematures can often be avoided by proper feeding, the early administration of iron, cod-liver oil and orange juice, more particularly in the artificially fed. The anemia resulting from nutritional disturbances can be prevented by a well balanced diet which includes prophylactic measures for rickets and scurvy. The instituting of vegetable soups and vegetable feeding as early as the sixth or seventh month, before the iron reserve is completely exhausted, is to be recommended.

The *treatment of the anemia proper* consists in arranging the *dietary,* providing the age of the infant permits, so that plenty of iron containing foods are given. Especially valuable are green vegetables, vegetable and

cereal broths, meat juices, and eggs. Bread and cereals should be limited so as not to crowd out the above. Fruit juices are also valuable. Spinach in powdered form can be added to the milk mixture to advantage once or twice daily.

Fresh air, sunlight, sufficient exercise and in general proper hygienic surroundings, are of tremendous importance. Hydrotherapy and massage may often be used advantageously.

Iron and arsenic play the principal rôle in the *medication*. A convenient form of iron medication is ferri et ammonii citrate, gr. ss to gr. iii, or the saccharated carbonate, grs. i to v, twice daily. In severer cases the hypodermic injection of iron, arsenic, or iron and arsenic combinations twice a week are recommended. The albuminates or peptonates of iron may be alternated with the inorganic salts where the treatment must be long continued. It should be remembered that there is a possibility of over-medication with iron and that the stomach may rebel when excessive dosage is administered. Liquor potassi arsenitis, in 1 to 3 minim doses three times daily, is a valuable adjunct to iron medication but the period of time over which it is administered should be limited. . Organotherapy and the administration of bone marrow have, on the whole, not yielded satisfactory results.

Blood transfusion often proves extremely valuable in the severe forms of anemia. Intravenous transfusion is the most satisfactory, but good results have followed intramuscular injections of whole blood. Transfusions should be repeated at regular intervals in severe cases. The most that should be expected from transfusion is a temporary benefit in the severe cases sufficient to bridge the period of shock and the time necessary to the therapeutic effect of other forms of medication. Prolonged exposure of the spleen to the rœntgen rays, with proper protection of the surrounding tissue, has been followed by marked improvement in a number of our cases.

In acute hemorrhage and some of the hemorrhagic diseases, physiological salt solutions, or better, Ringer's solution, intravenously or intramuscularly, may be indicated and in the former method of administration the addition of epinephrin to the solution is a valuable adjunct.

Splenectomy for cases of von Jaksch's anemia, with good results, are reported by Giffin[1] and Stillman.[2] Five operative recoveries with apparently lasting improvement out of six cases are reported by them. With a tendency to spontaneous recovery in von Jaksch's anemia, the advisability of subjecting young infants, generally, to this operation is open to question.

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[1] Giffin, H. Z.: Annals of Surg., 42, 676, 1915.
[2] Stillman, R. G.: Amer. Jour. of Med. Sciences, xxx, 153, 219, 1917.

Appendix.

PROPRIETARY BABY FOODS.

It should be borne in mind that the average daily cost of many of the proprietary baby foods is in excess of twenty-five cents.

For practical purposes the baby foods may be classed as follows:

GROUP I. Prepared from cow's milk.

1. Condensed milk without added sugar.
2. Condensed milk with added sugar (Borden's Eagle Brand) (F., 8.85; P., 7.34; milk-sugar, 11.61; cane-sugar, 42.9; ash, 1.77; water, 27.53).
3. Evaporated milk (St. Charles) (F., 9.0; P., 7.82; milk-sugar, 11.19; ash, 1.71; water, 69.91).
4. Peerless Brand unsweetened evaporated milk (F., 9.27; P., 7.28; milk-sugar, 9.99; ash, 1.51; water, 71.82).
5. Carnation Brand.
6. Lacta Præparata (powder).
7. Mammala (powder) (F., 12.12; P., 24.35; milk-sugar, 55.34; ash, 5; moisture, 3.19).
8. Dryco Brand powdered milk (F., 12.0; P., 34.0; milk-sugar, 44.0; ash, 7.0; moisture, 3.0).
9. Klim (Merrell-Soule Co.) Powdered milk—marketed as whole milk powdered, skim milk powdered, modified milk powdered.
10. Powdered albumin milk, Hoos albumin milk (protein milk), Merrell-Soule protein milk (albumin milk).

(425)

4. Denno's Baby Food (F., 1.79; P., 11.0; cane-sugar, 15.2; starch, 64.6; ash, 1.12; water, 6.2).

5. Allenberry's No. III (malted) (F., 1.05; P., 10.23; carbohydrates (sol.), 25.00; maltose, 16.5; dextrin, 8.5; carbohydrates (insol.), 60.01; ash, 0.60).

(C) Starch completely changed to dextrin and maltose:

1. Borcherdt's Dri-Malt Soup Extract (maltose, 71.10; dextrin, 13.50; protein, 8.66; ash, 2.94; moisture, 3.80). Calories per ounce by weight equals 110. It is a laxative, and is easily digested because of the high maltose and potassium carbonate (1.1 per cent.) contents.

2. Borcherdt's Malt Soup Extract (protein, 6.40; maltose, 57.57; dextrin, 11.70; ash, 2.54; moisture, 21.79). It contains 1.1 per cent. potassium carbonate.

3. Borcherdt's Dri-Malt Soup Extract with Wheat Flour. Semi-liquid malt soup extract, to which gelatinized wheat flour has been added, and the whole dried. One ounce equals 110 calories.

4. Borcherdt's Malt Sugar (dry) (maltose, 87 per cent.; dextrin, 5 per cent.). One ounce equals 120 calories. The following table will give a comparative idea of the relative value by weight and measure of Borcherdt's liquid and dri-malt soup extracts:

16 Fluid oz. equal 19.5 oz. dry malt powder by measure.
1 Fluid oz. equals 1.2 oz. dry malt powder by measure.
1 Ounce of liquid by weight equals 0.83 oz. of powder.
1 Fluid oz. represents 90 calories.
1 Ounce of powder by weight represents 110 calories.

5. Horlick's Malt Food (contains no milk) (F., 1.40; P., 12.06; carbohydrates (chiefly maltose), 81.97; ash, 2.60; water, 1.97. Calories, 109.29. It contains 1 per cent. of potassium bicarbonate.

6. Mellin's Food (F., 0.16; P., 10.35; maltose, 58.88; dextrin, 20.69; carbohydrates (sol.), 79.57; salts, 4.3; water, 5.6). Calories, 91.43. It contains 2 per cent. of potassium bicarbonate.

7. Dextri-maltose (Mead's No. 1) (maltose, 52; dextrin, 41; water, 5; sodium chloride, 2). No. 2 (maltose, 53; dextrin, 42; water, 5). No. 3 (maltose, 52; dextrin, 41; water, 5; potassium carbonate, 2).

8. Nahrzucker (Sohxlet) (F., 0.03; P., 0.13; maltose, 41.0; dextrin, 53.3; ash, 1.7; water, 2).

GROUP IV. Dry casein.
  1. Larosan (Roche), (calcium caseinate).
  2. Casec (Mead), (calcium caseinate).
  3. Protolac (Dry Milk Co.), (calcium caseinate).

GROUP V. Diastatic ferments.
  1. Diastoid (Horlick's, powder). Maltose 72.91 per cent.
  2. Diazyme (Fairchild, liquid), a good product.

GROUP VI. Peptonizing powders.
  1. Peptogenic milk powder (Fairchild's).
  2. Pepsin.

GROUP VII. Rennet powders (precipitating curd in a finely divided form).
  1. Chymogen (rennin and milk-sugar).
  2. Pegnin (rennin and milk-sugar).

GROUP VIII. Powdered vegetables.
    Carrot (Beebe).
    Spinach (Beebe).

It will be noticed that there are two great classes of proprietary infant foods:

THE FIRST. (GROUPS I, II). Those containing cow's milk.

Sweetened Condensed Milks. These are advertised as complete infant foods. All of them are quite similar in composition. All contain large amounts of cane-sugar. It is impossible to make, by simply adding water, a properly balanced food for an infant's continuous diet. A dilution to give a rational amount of proteins and fats has a large excess of sugars, and one to contain any amount under 7 per cent. total sugar would be so weak in both protein and fat that the baby's proper growth would be very seriously interfered with.

Eagle Brand condensed milk contains: fat, 8.85; proteins, 7.34; milk-sugar, 11.61; cane-sugar, 42.90; ash, 1.77; water, 27.5.

TABLE.

A Well-known Condensed Milk, Showing the Content of Various Dilutions. Fats and Proteins Deficient.

	Full strength Per cent.	6 parts water Per cent.	12 parts water Per cent.	18 parts water Per cent.
Fat	6.94	.99	.53	.36
Proteid	8.43	1.2	.65	.44
Cane-sugar	50.69	7.23	3.90	2.67
Salts	1.39	.17	.10	.07
Water	31.30	90.49	94.80	96.46

The Unsweetened Evaporated Milks. They are made by heating the milk to 200° F., and then transferring it to vacuum pans, where it is maintained at a temperature of 125° F., until sufficient water is evaporated to bring the product to the required condensation. In most products this milk is about double strength.

The sugar content not being in excess, these milks can be so diluted that a reasonable amount of fat and protein can be obtained, with, however, a considerable deficiency in sugar; this relatively low amount of carbohydrate can then be made up by adding sugar (cane or maltose-dextrin compounds), much the same as is done with cow's

milk. Where it is impossible to obtain clean, fresh milk, evaporated milk can be used with good success as a temporary diet in traveling, etc. A fresh can should be opened daily. It can be diluted with three to six or more parts of water, or cereal water and sugar in some form as indicated; however, the carbohydrates contained in the formula should rarely exceed 7 per cent. One part of milk to two parts of diluent plus carbohydrates is the strongest formula in which it is ever necessary to feed infants, as this equals the strength of whole milk with carbohydrate added.

Occasionally, infants with a very weak digestion will thrive on the evaporated milk where all other methods fail, if the food is started in high dilution, the quantity being increased as the infant shows improved capacity.

Because of the repeated heating and the low salt content, the food necessarily loses some of its vital requirements, and an early attempt to change to fresh milk should be made in order to avoid constitutional disorders as rachitis, scurvy, etc. The tendency to become very fat on this class of foods is proverbial, but this is not usually associated with high resistance or immunity to infections, and these infants succumb rapidly to the respiratory and intestinal infections. Unless the mother is forewarned, it is often with reluctance that she can be made to foresee the necessity of taking her baby off the food which agrees with it, and experiment with a new and occasionally uncertain formula.

The Powdered Milk Foods. Mammala, Honor Brand, and Merrill-Soule Brand are fresh milk dried. In the two former, part of the cream has been removed. All have some lactose added. They find their most important indication as an occasional substitute feeding in breast-fed infants—first, for the mother's convenience, to allow her recreation; secondly, where the milk of the mother is insufficient, and one or two regular feedings are indicated temporarily until a formula of fresh milk

is advisable, or while traveling, when the milk supply is uncertain; and thirdly, those containing large amounts of maltose (Horlick's) can be given once daily in breast-fed infants in need of a laxative.

THE SECOND CLASS. Those to be used in conjunction with fresh cow's milk. In this class belong GROUPS III and IV. These give us a far more rational infant food.

GROUP III. (A) The unchanged or partially dextrinized starches are especially to be used in solution in place of boiled water as diluents, best after the second month. A number of good cereal flours can be purchased on the market.

(B) In this group are found most of the highly advertised and detailed baby foods. They have little or no advantage over the plain cereal flours.

(C) These are especially valuable where maltose and dextrin are better taken than cane- or milk- sugar. Dextri-maltose (Mead's No. 1 and 2) and Nahrzucker.

DIRECTIONS FOR THE PREPARATION OF INFANT'S FOODS.

Tea.

To a small half-teaspoonful of fennel, chamomile, or "green" tea add 1 pint of boiling water, cover with a clean dish, and steep for two or three minutes, or till the tea is of a light yellow color; then pour through a clean sieve or muslin. It should be weak. If used for thirst only, in diarrheal cases, one-fourth of the above amount is sufficient.

Barley Water.

Soak 2 tablespoonfuls of washed barley (pearl) in water overnight; pour off water, add 1 quart of fresh water, and boil down to 1 pint (2 hours). Add boiled water to make 1 pint, if necessary. Strain through fine cloth. Keep in ice-chest.

Oatmeal and Rice Water.

They are prepared in the same manner, only boiled more slowly. They may be made from barley, oatmeal, or rice flours by using 1 rounded tablespoonful to 1½ pints of water, and boiling for 20 minutes down to 1 pint, in an open stew-pan, stirring constantly. (Approximates 3 calories per ounce). *In preparation of a feeding formula they can be prepared in a more concentrated form if indicated.*

Farina, Cream of Wheat, Oatmeal and Rice Flour Gruels.

To make six ounces use—1 tablespoonful cereal, ½ cup water, ½ cup milk, 1 pinch salt. Boil for thirty minutes over the direct flame or 1½ hours in the double boiler.

To Dextrinize Barley or Oatmeal Water.

Cool to 105° F., add 1 teaspoonful extract of malt, cereo, liquid taka-diastase or diazyme, stir, allow to stand for 15 minutes, when the gruel becomes thin and watery. Add a pinch of salt, stir, only to mix, cool, strain, and put in ice-chest.

Flour Ball.

Tie 2 pounds of wheat flour in a cheese-cloth bag, and boil in 2 quarts of water for five hours. Remove from water; place in oven until quite brown on the outside. This will require from two to three hours slow baking. Break open and throw away the brown shell; the remainder, the baked flour, must then be grated into a powder, or may be ground in a mill.

Albumin Water.

To ½ cup of cold boiled water add the white of 1 fresh egg and a pinch of salt. Stir very thoroughly. A piece

or two of artificial ice may be added before stirring. One-half teaspoonful of sugar and orange juice may be added, if not contraindicated. Barley water may be used.

Albumin Water with Beef Extract.

One-quarter teaspoonful of beef extract may be added to the cold water before adding the egg albumin.

White of Egg and Digested Gruel.

Whites of 2 eggs may be added to 1 pint of dextrinized barley, oatmeal, etc., gruels. Stir thoroughly.

Pasteurization of Milk Without a Thermometer.

Place milk as it comes from the dairy (with stopper removed and plug of sterile cotton inserted) in a pan of cold water with folded napkin beneath the bottle to prevent unequal heating. Let the water boil for three minutes. Allow to remain in hot water for eight minutes. Cool.

Pasteurized Milk (double boiler).

Place milk in cold water bath, having water to level of milk; bring milk to temperature between 155° and 167° F. for 15 to 20 minutes.

Sterilized Milk (double boiler).

The milk mixture is put into the inner vessel cold, and the water in the outer vessel is also cold. The double boiler is then placed on the stove and allowed to remain until the water in the outer vessel boils for 6 to 8 minutes; the whole process requires 10 to 15 minutes. While the milk heated in this manner forms a much finer and softer curd than that of raw milk, it is not as fine as the milk boiled directly over the flame.

Whey.

Heat 1 quart of clean raw milk to 104° F., and add 1 level teaspoonful of chymogen or fresh essence of pepsin (Fairchild's). Allow it to stand for one-half hour, pour off the free whey, pour the curd into a straining cloth for one-half hour, and collect the remainder of the whey.

Chymogen Milk.

Boil milk for five minutes, cool to 104° F., and add 1 full teaspoonful of chymogen to each quart of milk, and stir for one-half minute. Let it come to a clabber by allowing it to stand for 15 minutes; then beat it well until the curd is finely divided. Do not heat above 100° F., when preparing individual bottles for feeding, otherwise curds will clump, and will not pass through the nipple.

Indications for chymogen milk: (1) Vomiting in infancy; (2) indigestion due to the large curd formation.

Buttermilk in the Home.

A pure culture of lactic acid bacilli is added to raw, pasteurized, or boiled milk in an earthenware dish, and allowed to stand at about 80° F. for 15 to 20 hours, or until the casein is coagulated. Stir vigorously in a churn, or with a spoon or egg-beater until the curd is very small, and then push the contents through a fine wire strainer with a spoon. If the buttermilk is too thick, add a small amount of water. When the buttermilk is once made, a small portion (about 4 ounces) may be used as the inoculating agent for the next supply to be made. In this way the original culture may be made to last from six to eight days. The quality and action of the product made will vary but little. Add 4 ounces of buttermilk to 1 quart of fresh milk, incubate, and follow the above outline. Sometimes the milk will not coagulate, although it may smell sour. Stirring with a spoon will often pro-

duce coagulation in a few minutes. The fat present will rise to the top, and when coagulated appears as a brownish-yellow scum, which may be removed before the curd is broken up. At the present time the market is flooded with tablets for the preparation of buttermilk, but one must hesitate before using them to prepare milk for a baby. A pure culture should be used, or one recommended by the physician. Whole or skim milk is to be used as indicated in each individual case.

Startoline.

Carefully pasteurize 2 quarts of fresh whole milk to a temperature of 180° F. for one hour, or boil for five minutes; cool quickly to about 80° F., and add 1 ounce of Hanson's Lactic Ferment Culture, and let it stand undisturbed until well curdled, which should be in 15 or 20 hours, at a temperature of 75° F. Then place on ice. When ready to use, beat curd up with a spoon until it is of a creamy consistency.

Buttermilk for Hospital Feeding.

Pasteurize whole sweet milk to a temperature of 180° F. for one hour; then place in cold water until cooled to 80° F. Add 1 ounce of startoline to every quart of milk, stir with a spoon, and cover; allow to stand from 15 to 20 hours, then churn for one hour; then add a little cold sterile water to break butter away from milk; and strain buttermilk.

Buttermilk and Skim Milk Mixture.

To a few tablespoonfuls of buttermilk or skim milk add 2½ level tablespoonfuls of flour (flour ball or dextrinized barley flour), to make a paste. Make up to 1 quart with buttermilk. (1) Bring to a boil, withdraw from fire. (2) Bring to a boil, withdraw from fire a second time. (3) Add 4 level tablespoonfuls of cane-

sugar, and bring to a boil for the third time. (Maltose-dextrin preparations are best in all diarrheal conditions.) (1, 2, and 3 should require about twenty minutes time.) Make up to 1 quart with boiled water, if it has boiled away; put on ice. It is well to start with one-half the amount of sugar, and increase as indicated.

Indications for buttermilk and skim milk mixtures:

1. Fat indigestion.
2. Loose bowels (it may be necessary to reduce the amount of sugar. The high protein contents tend to constipate).
3. Malnutrition, with stationary weight..

Brady's Mixture No. 1.

Dr. Jules Brady, of St. Louis, has suggested the two mixtures following, which contain less carbohydrates than the above buttermilk mixture, and which he has found especially valuable in the feeding of infants in institutional practice.

Mixture No. 1, which is used for young infants during the first two months, contains 11 calories in each ounce; the young infant receives 4 ounces of this mixture for every pound of body weight as soon as it will take it. The baby weighing 6 pounds at birth is allowed to take 24 ounces in twenty-four hours, or 3.5 ounces every three hours, 7 feedings in twenty-four hours. The average infant at three or four days will take 1 ounce; at eight days, 1 to 2 ounces; at fourteen days, 1½ to 2 ounces; at three weeks, 2 ounces; at six weeks, 3 ounces; at eight weeks, 4 ounces.

Mixture No. 1.

¾ quart buttermilk or skim milk.
¼ quart barley water (thick).
1 ounce by measure, Mellin's Food.
½ ounce granulated sugar.

The ingredients are mixed together in the following manner: To the barley gruel is added the cane-sugar and the Mellin's Food, and then the milk is slowly added, and the mixture strained. As a rule, the milk is acidified with lactic acid bacilli twelve hours before being made up, having first agitated it.

Brady's Mixture No. 2.

On reaching a weight of 8½ to 9 pounds, infants receive the mixture No. 2, which contains 18 calories for every ounce. The babies are allowed 3 ounces of the mixture No. 2 for every pound of body weight.

Mixture No. 2.

⅔ quart whole fresh milk or whole buttermilk.
⅓ quart barley water (thick).
1 ounce granulated sugar.

Keller's Malt Soup.

To 11 ounces (330 Gm.) of warm milk gradually add 1⅔ ounces (50 Gm.) of flour, stir constantly, then pour through a clean sieve or muslin. In another dish dissolve 3 ounces (100 Gm.) by weight, or 2½ ounces or tablespoonfuls by measure, of Borcherdt's malt extract with potassium carbonate in 20 ounces (600 Gm.) of boiled warm water. Then mix both solutions, put on fire, stir continuously, and boil for two or three minutes.

Indications for Keller's Malt Soup:
1. Fat indigestion.
2. Disturbed metabolic balance (fat-soap stools).
3. Chronic constipation (often relieved by simple addition of malt soup extract to ordinary milk mixture in place of part of sugar).

Contraindications:
1. Before the third month, if the stools are loose.

2. For a period of more than four to eight weeks (to be followed, where possible, by ordinary milk mixtures, the strength of the latter being gradually increased).

Cream Soups.

Cream soups may be made from vegetable pulp, using 1 tablespoonful of cooked potatoes, peas, or asparagus to ½ cup of water in which the vegetables were cooked, ½ cup of sweet milk, and ½ teaspoonful of flour, with a little butter and salt. Cook another minute or two. Strain if necessary. Serve.

Corn or tomatoes may be used in the same manner, using 2 tablespoonfuls of strained vegetables, with about one-third water and two-thirds milk. When tomatoes are used, add a small pinch of soda to tomatoes before adding other ingredients.

Vegetable Soup.

One-fourth pound lamb stew, cut into pieces, 1 potato cut into pieces, 1 carrot cut into pieces, 2 stalks of celery cut into pieces, 1 tablespoonful of pearl barley, 2 tablespoonfuls rice, 2 quarts water. Boil down to 1 quart; boil three hours. Add pinch of salt, and strain before feeding.

Lamb, or Veal Broth.

Lean meat chopped fine, 1 pound; cold water, 1 quart; a pinch of salt; cook slowly two or three hours to 1 pint. Add water from time to time, so that when finished there will be 1 pint of broth. Strain; when cold, skim off fat.

Chicken Broth.

Small chicken, or one-half of large fowl, with all skin and fat removed; chop bones and all into small pieces; add 1 quart boiling water and a little salt; cover closely, and

allow to simmer over a slow fire for two hours. After removing allow to stand one hour; then strain. Add water, if necessary, from time to time, so that there will be 1 pint when finished.

Farina Soup.

To 1 pint of meat broth, gradually add, while stirring, 1 even tablespoonful of farina, and boil down to 1 cup (½ pint) in about twenty minutes. It is a good plan to boil the farina for from fifteen to twenty minutes before adding it to the broth; then broth and farina need to be boiled together for but ten minutes.

Dried Fruit Soup.

Wash thoroughly 1 cup of dried apricots and 1 cup of prunes. Cook in 1 quart of cold water until very soft. Strain and press out all juice. Sweeten to taste. Thicken with a tablespoonful of rice flour to 1 quart of the liquid. Cook twenty minutes to remove the raw taste of the flour.

Soy Bean and Condensed Milk (Ruhrah).

Add a level tablespoonful of soy bean flour to 2 level tablespoonfuls of barley flour, add a pinch of salt, and mix to a paste with boiled water, adding further water to 1 quart. Boil for twenty minutes, and add water to make up for the loss due to evaporation during boiling, so that total mixture is 1 quart. Condensed milk is now added, varying in quantity from ½ to 1 dram of condensed milk to each ounce of the mixture, depending upon the age and the condition of the infant. Double the quantity of soy bean and barley flours may be used for older children. Each ounce of soy bean gruel contains 10 grams of protein and 102 calories. Two ounces of soy bean gruel in a quart of water contains 0.56 per cent. protein, 0.62 per cent. fat, and 3.31 per cent. sugar.

The quantity of the feedings may be varied according to the condition and needs of the infant, varying from 1 to 8 ounces per feeding.

It is indicated whenever fresh clean milk is not obtainable, in infants with marasmus, in some intestinal disturbance associated with diarrhea.

Beef Juice.

Take ¼ to ½ pound round steak, broil slightly, cut into small pieces, and then press out the juice with a meat press or potato ricer, and add a small pinch of salt. Feed fresh, or warm before giving, but do not heat sufficiently to coagulate albumin.

Potatoes.

Boil potatoes in salt water in the ordinary way until they are thoroughly done. Then mash through a very fine sieve, and add a little butter.

Spinach.

Cook spinach in salted water until tender. Pour cold water over it, and drain. Chop fine, or rub through a coarse sieve. To 2 tablespoonfuls of spinach add 1 teaspoonful of fine breadcrumbs, ½ teaspoonful melted butter, and a little salt. Reheat and serve.

Asparagus.

Cook one-half of a bunch of asparagus in about a pint of slightly salted water. When tender, remove stalks one by one. Place on a warm plate, and remove pulp by taking hold of the firm end of the stalk, scraping lightly with a fork toward the tips. Use pulp only. Make a sauce with one-fourth of a cup of water in which asparagus was cooked, one-fourth of a cup of milk, 1 teaspoonful flour, a little butter and salt. Dip a small piece of toast in the sauce. Take what is left of the

sauce and mix with 2 tablespoonfuls of asparagus pulp. Reheat. Place on toast and serve.

Carrots.

Cook ½ pound of young carrots in a pint of fat-free soup stock or slightly salted water, adding more if it cooks away before they are done. Rub through a sieve; add 1 teaspoonful of bread-crumbs, a little butter and salt. Reheat and serve.

Beans.

Soak 2 ounces or 4 tablespoonfuls of beans, and cook them slowly in a good deal of water until they are soft, but not broken. Rub through a sieve, add 1 cupful of soup stock, and let them cook for one-half hour, adding more stock if it boils away. Mix a little butter and flour, about a teaspoonful of each, and a little salt. Add to soup. Return to fire, and cook for a few minutes.

Green Peas.

Cook a cupful of green peas in boiling salted water until they are done. Drain, saving the water in which they are cooked. Rub through a coarse sieve. Make a sauce of 2 tablespoonfuls of water in which the peas were boiled, 2 tablespoonfuls of sweet milk, ½ teaspoonful flour, ½ teaspoonful fine bread-crumbs. Mix all together. Reheat and serve.

Fruits.

(a) *Orange Juice:* Take sweet orange, cut into halves, and squeeze out juice by hand or with a lemon squeezer; strain, put on ice, and use as ordered.

(b) *Prune Juice:* Take ½ pound of prunes, wash thoroughly, cover with cold water, and soak overnight. In the morning place on stove in the same water, and

cook until tender.   Add 1 teaspoonful of sugar, and strain.

(c) *Prune Jelly:* Cover 1 pound of prunes with 1 quart of water; cook slowly until tender; pit, and press pulp through a sieve. Add sugar to sweeten (2 teaspoonfuls) and ½ box of gelatin dissolved in a pint of water, and boil. Strain, cool, and keep covered.

(d) *Apple Sauce:* Take 6 apples and peel, core, and cut them into quarters. Place them in an enameled dish; sprinkle over them 1 tablespoonful of sugar; add 1 cup of cold water; put the dish on the stove, and boil the apples to a mush (about thirty minutes).

(e) *Orange Gelatin:* Soak ½ box of shredded gelatin in cold water for thirty minutes. Add 2 cupfuls of boiling water, and dissolve. Then add 1 cupful of sugar, the juice of 1 lemon, and a cupful of orange juice. Strain through a fine strainer (or a cloth) into moulds, and set away to harden.

Eggs.

Use only soft-boiled or poached eggs. Be sure that the eggs are fresh. Drop egg in boiling water; immediately turn flame out, and allow to stand for five minutes.

Casein Gruel.

To make 10 ounces—each ounce equals 18 calories: Casein, 6 level tablespoonfuls; flour ball, 2 level tablespoonfuls; water, 6 ounces; milk, 8 ounces.

Mix casein and flour ball together with the water— let boil three minutes directly over the flame, stirring constantly. Add the milk gradually and bring again to boil. Place in double boiler and cook three to four hours, stirring occasionally. Strain.

Thick Cereal (Sauer).[1]

Skimmed milk, 9 ounces; water, 12 ounces; farina, 6 tablespoonfuls; Dextri-Maltose, 3 tablespoonfuls.

Boil for one hour in a covered double boiler. Sugar and salt may be added.

Pap.

Put 1 pint of milk on to boil; add butter the size of a walnut. Beat 1 egg thoroughly. When milk boils, add the beaten egg, stirring constantly. Mix 1½ tablespoonfuls flour into a paste and add to mixture, stirring constantly. Allow mixture to boil ten minutes. Just before taking from the fire add a pinch of salt. May be taken plain, or with milk and sugar as directed.

Cornstarch Pudding.

Take 1 pint of milk and mix with 2 tablespoonfuls of cornstarch; cane-sugar, 1 tablespoonful. Flavor to taste; then boil the whole eight minutes. Allow to cool in a mould.

Custard Pudding.

Break 1 egg into a teacup and mix thoroughly with sugar to taste. Then add milk to nearly fill the cup. Mix again, and tie over the cup a small piece of linen. Place the cup in a shallow saucepan half full of water, and boil for ten minutes.

If it is desired to make a light batter pudding, a teaspoonful of flour should be mixed in with the milk before tying up the cup.

Infant's Gelatin Food.

About 1 teaspoonful of gelatin should be dissolved by boiling in ½ pint of water. Toward the end of the boil-

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[1] L. W. Sauer: Thick cereal in the treatment of pyloric stenosis. Arch. of Ped. xxxv, 385, 1918.

ing, ¼ pint of cow's milk and 1 teaspoonful of arrow-root (made into a paste with cold water) are to be stirred into the solution, and 1 to 2 tablespoonfuls of cream added, just at the termination of the cooking. It is then to be moderately sweetened with white sugar, when it is ready for use. The whole preparation should occupy about fifteen minutes.

Albumin or Eiweiss Milk (Finkelstein).

One quart. Take fresh whole milk, bring to a temperature of 98° to 100° F. Then add 2 level tablespoonfuls of chymogen powder to a quart of milk; place in a water bath of 107° F., for fifteen to twenty minutes, until coagulated. Then hang in a sterile muslin bag for one hour to drain.

To the curd of 1 quart of milk add 1 pint of buttermilk, and rub through a copper gauze strainer three times. Then add 2 level tablespoonfuls of wheat flour, flour ball, or Imperial Granum, rubbed to a paste with 1 pint of water. Boil ten minutes, cutting back and forth constantly, not stirring, with a large wooden spoon, otherwise large curds will form. If needed, water should again be added, to make the finished mixture one quart. Finkelstein advises the early addition of 3 per cent. of carbohydrate in the form of a maltose-dextrin compound. This is best done by dissolving the sugar in a moderate quantity of water, and adding while the mixture is being boiled. It must not be heated above 100° F. before feeding, otherwise it will clump.

Albumin milk contains: protein, 3 per cent.; fat, 2.5 per cent.; milk-sugar, 1.5 per cent.; starch, 1.0 per cent.; salts, 0.5 per cent. Caloric value is 450 calories per liter, or 12 calories per ounce.

Indications for albumin milk (Finkelstein):

1. Diarrheas and all cases of abnormal intestinal fermentation (sugar).

2. Fat indigestion with low sugar tolerance.
3. Gastro-intestinal infections associated with frequent stools.
4. Systemic infections with intestinal complications.

Albumin Milk (Müller and Schloss).

Use 1 quart of water and 1 quart of buttermilk, and boil for three minutes. Set aside for thirty minutes, and then pour off the upper 36 ounces of the whey. Boil the upper 4.5 ounces of a quart of fresh milk for three minutes. Add 1 ounce of dextri-maltose to the boiled top

Fig. 28.—Utensils needed for artificial feeding: Double boiler (small), pan, funnel, bottle-brush, 250-mil (8 oz.) graduated glass or pitcher, 6 nursing bottles and rack, paper caps for bottles (sterile), nipples, milk, sugar, flour, milk of magnesia, citrate of soda, tablespoon, dairy thermometer, vegetable mill.

milk, and to this add the curds from the first mixture, which would equal 27.5 ounces, making 1 quart of the milk mixture.

Larosan Milk.

Two-thirds of an ounce of Larosan powder (p. 428) is added to ½ pint of milk, and mixed thoroughly. Another whole pint of milk is heated to the boiling point. When it has come to a boil, it is added to the Larosan milk mixture, and the whole is placed on the flame and allowed to boil for five minutes. This may be diluted

with water in the proportion of one-half Larosan milk and one-half water, or two-thirds Larosan milk and one-third water.

This mixture, because of its high protein content and comparative ease of preparation, can be used as a substitute for albumin milk in the home.

Butter and Flour Mixture of Czerny and Kleinschmidt.[1]

The *Butter-flour feeding* which is recommended by Czerny and Kleinschmidt represents a utilization of the fat of the cow's milk. It is claimed that the mixture is well tolerated, even by very young and underweight infants.

For the preparation of this feeding, for every 100 Gm. of the diluting fluid, 7 Gm. of butter, 7 Gm. of wheat flour and 5 Gm. of cooking sugar are used. The butter is heated above a small flame, with good agitation with a wooden spoon until it begins to foam and all odor of fatty acids has disappeared. Then the wheat flour is added and mixed with the melted butter. Both of these are then cooked over a moderate fire (asbestos plate), with constant mixing until the mass becomes thin and somewhat brownish. Then 100 Gm. of warm water and 5 Gm. of sugar are added and the entire mixture is again boiled, strained through a hair sieve and finally added to the boiled and afterwards cooled milk. The mixture ready for feeding must be kept cool, but it is not necessary to sterilize it again. The amount to be added to milk mixtures in infants below 3000 Gm. should be one-third; in those of about 3000 Gm. and above, about two-fifths of the final mixture. It should be administered in quantities of not more than 200 Gm. per kilo body weight in a day.

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[1] Jahrbuch f. Kinderh. 87, 1918.

Meats.

Raw or slightly cooked beef, scraped and seasoned, can be fed in amounts equaling a tablespoonful at eighteen months or sooner, once daily.

Take meat, preferably from the round, free from fat. Place on a board and scrape with a silver spoon. When you have the desired amount of meat pulp, shape into a pat and broil on a hot, dry spider. Do not cook too long. When done, season with a little salt and butter. Serve. A few drops of lemon juice may be added.

Later, lamb, beefsteak, roast beef and chops are the best, and should be broiled. By no means fry any meat for the baby. Soup meat, well cooked, may also be given. All meats should be very finely cut before giving them to children.

BOTTLES AND NIPPLES AND THEIR CARE.

The nursing bottle should be of such a construction that every portion of it is easily reached with a proper brush. This necessitates the avoidance of sharp corners and angles, and makes the smooth stream lines in its construction desirable. It should be made of good glass, not easily broken, capable of being boiled repeatedly without cracking, and should hold about 8 to 10 ounces. Several nursing bottles should be kept on hand, and, if possible, as many bottles as there are nursings in a day should be available, so that the whole day's feeding may be prepared according to the particular formula, and the mixture then iced, and the individual bottles warmed on a water-bath whenever necessary. New bottles should be annealed by placing them in a vessel with cold water, and then bringing the water to a boil, boiling for twenty minutes, and then leaving the bottles in this water until it will cool off again. Bottles thus treated do not crack so easily when hot fluids are poured into them. After nursing, the bottle should immediately be rinsed with

cool water, and then washed with hot water and soap
suds by means of a bottle brush. Afterwards the bottle
should be set aside, inverted, so as to drain. Before use,
the bottles should be boiled for five minutes. To avoid
cracking, they must be placed in cold water and heated
slowly. After the food has been prepared, the individual
bottles may be filled and stoppered with sterile cotton, or,
better, sterile paper caps, which are sold for this purpose.
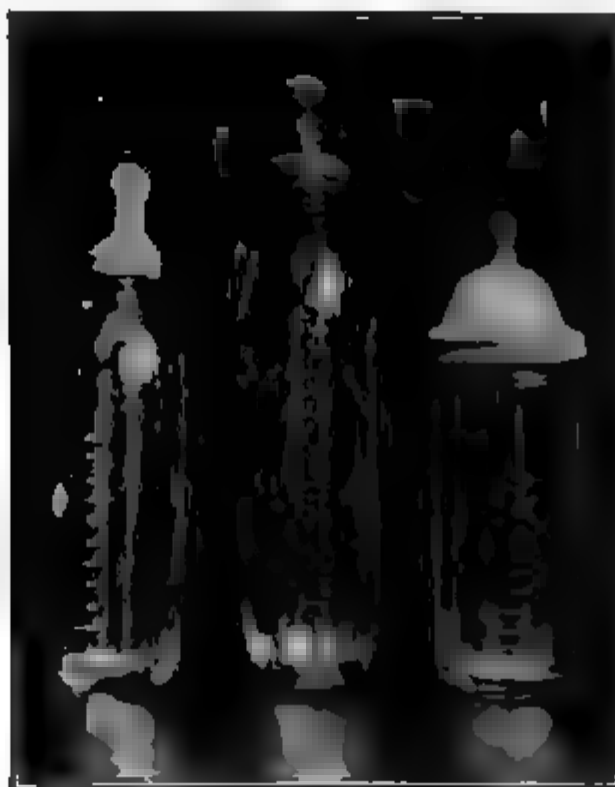
Fig. 29.—Good and bad nursing bottles. 1. Ordinary
small-neck nursing bottle as sold in drug stores (8-ounce).
2. Improved large-neck nursing bottle (made in 5- and 10-
ounce size). 3. Hygiea nursing bottle.

Nipples that can be turned inside out and easily
cleansed should be selected. The conical shaped nipple is
preferable. The hole in the nipple should be of such size
that the milk will drop rapidly and not flow when the
bottle is inverted. New nipples should be boiled before
they are used. After using, every nipple should imme-
diately be washed with soap and water, being turned in-
side out, boiled and finally dropped into a sterile jar,

where it is to be kept dry until ready for use again. Keeping the nipples dry lengthens the life of the rubber. Several nipples should always be kept on hand.

CARE OF FOOD DURING TRAVELING.

Whenever possible, the baby should be kept on its usual diet during the long journey. This is usually ac-

Fig. 30.—A milk station consisting of three rooms. Room 1. For all used bottles, bottle washers, and steam bottle sterilizers. Room 2. A clean room for preparation of formulæ. This room also contains milk separator, fat-testing apparatus and butter churn. Room 3. Pasteurizing and sterilizing apparatus.

complished without much difficulty when the baby is on boiled milk. If it has been fed on a raw milk mixture, the milk must be boiled before starting. When for any reason it is impractical to carry the milk mixture, evaporated milk or powdered milk may be used. (See Proprietary Infant Foods, page 425.) In the use of evapor-

ated milk, a fresh can must be opened at least once daily. When it is known that the baby's formula is to be changed, it should be tried out on the new food before starting on the journey. As soon as possible, the previous diet should be re-established. All water given to the baby while traveling must be boiled. The infant's food, after boiling for at least ten minutes, should either be placed in individual nursing bottles, or in bottles holding not more than 1 pint, so that not more than two or three feedings should be given from a single bottle. The bottle should be packed in ice, using care so that none of the ice reaches the top of the bottle. Upon reaching the train they should be placed in the ice-box of the dining or buffet car, unless a private ice-box is available. The baby's bottle can be warmed on the train by setting in a dipper of warm water, which may be carried hot in a thermos bottle, if the journey is to be a short one. Care must be taken that the water be not too hot, otherwise the cold bottles will be cracked. The nipples may be carried in a wide-mouthed, well-corked bottle, sufficient to cover the individual feedings. The nipples and bottles should be cleansed immediately after use.

THE DIAPER.

The diaper should be made of soft, light, and absorbent material, such as cotton diaper cloth, which can be purchased for this purpose. Cotton-flannel is too little absorbent, and soon becomes hard as a result of washing. A second diaper may be folded into a square, and be laid under the hips to prevent the moisture from reaching the clothes, or instead of this arrangement, which is rather heating and bulky for summer use, a small diaper may be folded two or three times to form a square of about nine inches, and this may be placed inside of the larger diaper to receive the urine and feces. About four dozen diapers are needed for an average baby.

A rubber or waterproof cover should never be applied outside the diaper. It is very heating, and liable to produce chafing and eczema. Diapers should be changed as soon as soiled, except at night, when they should be changed when the child is awakened for feeding, or when it is awakened by its own discomfort. Soiled diapers are always a source of discomfort, and not infrequently the cause of severe irritation of the skin, as well as of infections of the genital and urinary tracts. This is especially true in the case of female infants. No diaper should be applied a second time without first being washed. All diapers which have been soiled by discharges from the bowel should have the bulk of the feces removed from the diaper, and should be immediately washed with soap not too alkaline in character, and later boiled for twenty minutes, and thoroughly rinsed, so that all alkali may be removed. They should then be aired thoroughly. Soda and washing-powders should be avoided because of the danger of irritating the child's buttocks after being moistened by the urine.

The diapers of an infant ill with an intestinal infection should be cared for separately from those of other children. After changing the diapers, the nurse's hands and nails should be scrupulously cleansed with brush and file.

BABY'S DAILY BATH.

The baby should be bathed at least once a day, and on hot days even as many as three sponge-baths may be given. In the first six months the temperature of the bath should be 100° F., and in the second half of the year from 90° to 95° F. The room in which the bathing is done should have a temperature of at least 70°, and not more than 75° F.

Toward the end of the first year the infant may be sprayed for 15 to 30 seconds with water at 75° to 80° F. This should be followed by brisk rubbing of the entire

body. In young infants the bath is most conveniently given before the mid-morning feeding, and the face and hands may be sponged before the 6 o'clock feeding. In older infants, a cool sponge and massage may be given in the morning, and the warm bath at bedtime.

Before the umbilical cord has separated, sponge-bath only should be given, and never a submersion bath, for

Fig. 31.—Hospital bathroom. Located between two small wards for infants, showing two metal water jackets resting on a porcelain sink. These can be filled with water, and have a registering thermometer for indicating the temperature before giving the bath. They are covered with a clean towel for each baby. Baby is showered from an automatic mixing tank, which registers temperature of the water in the tank. The room further contains a scale and a low dressing table, with the various dressings, powders and ointments to be used. Also low nursery chairs, collapsible bags for soiled linen, and waste basins.

the fear of infection of the umbilical stump. Sponge-bath may be given on a towel, and when a tub-bath is

given, the child should be allowed to rest upon the attendant's left arm, which is slipped under its back from the baby's right side. By grasping the baby under the armpit with the left hand a good hold is secured, which prevents slipping. The right hand is left free for washing the baby. A special wash-cloth, preferably of cheese-cloth, should be provided for washing the baby's face and head.

A pure, bland, white soap should be used. Very little soap is needed for cleansing the baby's skin, and it is most important that the skin should be thoroughly rinsed. If the skin is sensitive and easily irritated, soap should be avoided, and the bran-bath (made by putting a handful of bran in cheese-cloth bag and soaking this in the water until milky) should be used.

After the bath the baby should be wrapped in a large soft towel and dried by sponging, and not by rubbing. Special attention should be paid to folds and creases of the skin, and these should be well powdered after being thoroughly dried.

Only warm baths should be used in infants who become pale and cyanotic when a cooler bath is used.

Care should be taken in bathing all children suffering from coughs. Great care should also be used while bathing a child suffering from vulvovaginitis, to avoid infection of the eyes.

COLD BATH AND COLD PACK.

Cold bath is an efficient antipyretic and nervous depressant in cerebral irritation, but it is a somewhat severe procedure for the infant, and is less frequently indicated than in the adult. It is to be used only in infants who react well. The bath is started with water at 100° F., and the temperature is then gradually lowered by the addition of ice-water, down to about 80° F. The infant should be continually rubbed while in the bath. The

bath should not be longer than five to ten minutes, and should be discontinued at once, if any cyanosis appears. The infant must be dried quickly, and then wrapped in a dry blanket, without dressing, and put to bed.

In most cases, however, a cold pack is preferable to cold bath, especially in young infants, as the former is a somewhat milder procedure. Cold pack is one of the best antipyretic procedures in infancy and childhood. The naked child is wrapped in a blanket wrung out of water at a temperature of about 100° F., and is then rubbed with ice through the blanket for about five to ten minutes. Ice-bag to head and hot-water-bag to feet are very useful—often necessary. After rubbing with ice, the child is left in the blanket, and covered well. The blanket may be removed, the child dried; and put into a dry blanket after about one hour.

HOT BATH.

Hot bath is indicated in cases of collapse or shock as a stimulating procedure; and prolonged hot bath as a diaphoretic procedure. It should. be started with water at a temperature of 100° F., and the temperature gradually raised to about 105° F. by addition of hot water. An ice-cap or cold cloth should be applied to the head. A thermometer should always be used while giving a hot bath. The infant should be well rubbed during the bath, which should be continued for about ten minutes. After the hot bath the infant should be well dried, until the skin is red, and then wrapped in a blanket and put to bed.

MUSTARD BATH AND MUSTARD PACK.

Mustard bath and mustard pack are indicated for their stimulating effect in cases of shock, or collapse, and in acute congestion of internal organs, and also in convulsions.

The amount of mustard used and the temperature of water is the same in both procedures. Powdered mus-

tard, in quantity of about 1 level tablespoonful to each gallon, or 1 teaspoonful to each quart, when smaller quantities are sufficient, should be used. Full quantity of mustard powder is first dissolved in about a gallon of warm water, and to this the rest of the water is added, while preparing the bath. For giving the pack, a smaller quantity of water is usually required. The. temperature of the water should be about 100° F., and it may be raised to about 105° F. by addition of. hot water. Cold applications should be made to the head.

The bath should be continued for about ten minutes, accompanied by rubbing the skin, and followed by ablution with lukewarm water, rapid drying, wrapping in a blanket, and rest.

Mustard pack is somewhat less efficient than mustard bath, but it is also less severe and less disturbing to the infant. The naked child is wrapped in a blanket which has been wrung out of water prepared as above stated. The infant is left in the pack until the skin is well reddened—about ten to twenty minutes—then washed off with warm water, followed by lukewarm water ablution, dried, and put to bed without dressing.

STOMACH WASHING.

The apparatus for stomach washing consists of a soft rubber catheter, 20 to 24 French, or infant stomach-tube, a small funnel, attached to a rubber tube, and a glass connection between the catheter and the tube.

The infant is wrapped with the arms confined, and is held in the sitting position, with a large basin at the nurse's feet. The tongue is depressed with the forefinger of the left hand, and the right hand passes a catheter rapidly backwards into the pharynx and down into the esophagus. Gagging is aggravated by passing this catheter slowly. After the catheter is part way in the esophagus, it should be passed more slowly. As the

cardiac orifice is passed, and the catheter enters the stomach, gagging again becomes more evident. This can be used as a sign that the catheter is entering the stomach. A good rule to follow in passage of the catheter is to measure the distance from the root of the nose to the tip of the ensiform cartilage, which approximates the distance from the teeth to the cardiac end of the stomach, and then pass the catheter about an inch farther. The passage into the stomach is usually marked by the appearance of curdled milk in the glass connecting tube. The funnel should now be raised as high as possible, to facilitate the escape of any gases from the stomach, and should then be lowered, in order to siphon any fluid contents. The funnel is then raised, and warm water at a temperature of about 100° F. is poured into the stomach quickly. The amount of water passed into the stomach at any time should about equal the quantity of the feeding to which the child is accustomed. The funnel is then lowered, just before all of the water leaves the tube, and the water siphoned out. This procedure is repeated a number of times, until the fluid comes back clear. During withdrawal, the tube must be compressed carefully to prevent leakage into the larynx. The washings should be collected and measured, so that the quantity remaining in the stomach may be estimated.

Sterile water or one-half strength normal saline, Ringer's solution, or a solution containing sodium chloride 5 Gm., sodium bicarbonate 5 Gm., and water 100 mils, may be used. It is frequently advisable to allow part of the solution to remain in the stomach.

Stomach washing is indicated in vomiting due to pylorospasm, hypertrophic pyloric stenosis, all forms of gastric irritation, chronic indigestion, acute dilatation of the stomach, and food and drug poisoning.

CATHETER FEEDING BY MOUTH.

The same apparatus is used as in stomach washing, the same technic being used for the introduction of the catheter, except that its tip should not be made to pass the cardiac end of the stomach, the food being allowed to enter the esophagus just above the cardia. This is accomplished by passing the catheter about one-half inch less than the distance from the root of the nose to the tip of the ensiform cartilage. The infant should be lying on its back, and not in sitting posture, as recommended in stomach washing. When the feeding is finished, the catheter should be tightly pinched between fingers and rapidly withdrawn, to prevent any food from trickling into the larynx. It is often advisable to wash the stomach before the food is introduced. (See Fig. 7, page 89.)

Catheter feeding is indicated in the feeding of premature infants, infants refusing their diet, those too weak to nurse, in the presence of persistent vomiting, and in all cases of delirium and coma.

CATHETER FEEDING BY NOSE.

This is not indicated in young infants. In older children it is often impossible to pass the catheter through the mouth, without undue struggling. It is also indicated in throat paralysis following poliomyelitis and diphtheria, and after throat operations and intubation. The method is similar to that described in catheter feeding by mouth, except that a smaller catheter (No. 15 French) is to be used.

IRRIGATION OF THE COLON AND RECTAL FEEDING.

The apparatus varies somewhat with the purpose to be accomplished. Where large quantities of fluids are to be introduced, it is necessary to use a douche-can or

fountain syringe, 4 to 5 feet of tubing, and a flexible
rectal tube or soft rubber catheter (size 20 to 24 French).
When small quantities are to be introduced, a glass fun-
nel may be used in place of the douche-can. When large
quantities of fluid are used, the can must not be raised
more than 2 feet above the child's body. The child
should be turned upon its side, with the lower limb ex-
tended, and the upper thigh flexed upon the abdomen.
The catheter should be well oiled, and introduced for
about 3 to 4 inches when large quantities are to be given,
and further introduction of the catheter may be made
while the solution is flowing into the rectum. For most
purposes the solution should be about 100° F.

Indications. 1. To produce evacuation of the bowel.
A salt solution containing a level teaspoonful of salt to
a pint of tepid water or weak soap-suds solution, or a
teaspoonful of glycerin in an ounce of water; or in the
presence of large fecal masses, 2 or 3 ounces of sweet
oil may be used.

2. To reduce temperature. At least 1 to 4 quarts of a
salt solution or weak soap-suds enema at about 95° F.
should be used, allowing about ½ to 1 pint to enter the
rectum, and repeating after expulsion.

3. Rectal feeding. A normal salt solution or nutrient
enemata containing 2 level tablespoonfuls of dextrose to
the pint of normal saline solution may be used. It is
indicated in cases of acidosis, and also in the presence of
vomiting, intoxication, and decomposition where the body
is in need of water. It is usually necessary that only a
small amount (2 to 6 oz.) of this solution be introduced
at a time, or that it be given by the drop method. Other-
wise it will not be retained. It should be repeated at
regular intervals of from two to four hours. It may be
necessary to compress the buttocks for twenty minutes
after administration, when the fluid is not well retained
otherwise.

4. Medication. There are two indications for rectal medication: (1) For the systemic effect. The drugs most commonly used for this purpose are chloral hydrate and the bromides, more especially in the presence of convulsions or coma. They should be diluted in small quantities of water or salt solution, not over 1 ounce, and may be administered in about four times the oral dose for the given age. (2) For local effect. Enemata are indicated for their local effect in the presence of marked tenesmus, inflammation, ulceration and hemorrhage. Not infrequently the tincture of opium (3 to 5 drops) and tincture of belladonna (3 to 5 drops) are administered, probably best in a 10 per cent. starch solution, for their sedative effect. In the presence of inflammatory processes, 1 per cent. silver nitrate solution may be used.

SALINE SOLUTIONS.

Solutions administered subcutaneously and intravenously should be maintained at a temperature approximating 100°F.

1. For subcutaneous use. They are especially indicated in the presence of considerable loss of body fluids through vomiting, refusal of diet, and diarrhea, and in the presence of acidosis. Rectal administration should first be tried, and, in case that sufficient fluids cannot be administered to meet the infant's needs in this way, hypodermoclysis should be instituted. In infants 2 to 4 ounces can usually be administered, and in older children 4 to 6 ounces. This can be repeated every four hours, if necessary, or until fluids can be supplied by another route. Fluids can be administered beneath the skin of the abdomen, chest, or lumbar region. There is some shock accompanying the administration of large quantities of fluids subcutaneously, probably due to the pain, and it is frequently necessary to give a child in collapse some subcutaneous stimulation of camphor in oil (10 per

cent. 1 mil), or adrenalin solution (1:1000, about 5 drops), before administration. The stimulating injection is to be made in regions of the body other than where the saline injection is made.

The best solutions for this purpose are

    (a) NaCl .... 7.5 grams.
          KCl .... 0.1   "
          CaCl .... 0.2   "
          Water, ...q. s. ad 1000.0 mils.
    (b) Dextrose may be added to the above solution in proportion of 50 grams to the liter (5 per cent.).

All solutions used for subcutaneous administration should, if possible, be made from fresh distilled water, and re-sterilized shortly before use.

2. Intravenous injections. The same solutions as indicated for subcutaneous use may be administered intravenously. Sodium bicarbonate, 30 Gm. to the liter, being added in the presence of acidosis and dextrose, 50 Gm. to the liter in cases of malnutrition and decomposition. Either direct or indirect transfusions of blood are also of extreme value in the presence of marked marasmus.

Technic. In older infants and children the injection may be made into the external jugular or median basilic or median cephalic veins. In young infants with open fontanelle, the longitudinal sinus is the most convenient point for administration. However, in the use of the latter method extreme care must be used, because of the ease with which the sinus wall can be punctured. All apparatus used in the intravenous administration must be thoroughly and freshly sterilized before use. Where a moderate quantity of fluid is to be administered (2 mils, 10 mils, or 20 mils) all-glass Record or Luer syringes can be used. In injection of fluids into the longitudinal sinus a short bevelled needle, about 0.75 inch in length, should be introduced at the posterior angle of the fontanelle.

The region of the fontanelle is sterilized, and the first syringe is three-quarters filled with the fluid to be in-

jected. The syringe is now connected with a needle by means of a short piece of rubber tubing to allow flexibility in case of movements on the part of the child, and the needle is passed into the sinus, its entrance being recognized by a sudden lessening of the resistance. Helmholz[1] suggests that the question of negative pressure within the sinus is one that must not be overlooked, and it is always well in entering the sinus to have the syringe attached, and before injection to withdraw blood, to make sure that the needle is in the sinus. Unless a head-clamp, as described by Helmholz is available, two assistants are required, one to hold the child's head firmly, and the second to manage the syringe, while the physician steadies the needle. From 100 to 200 mils of either a saline, dextrose solution or citrated or fresh blood can usually be administered without difficulty. Unger[2] has described an apparatus whereby large quantities of fresh blood can be transfused.

HOME-MADE ICE-BOX.

The following home-made ice-box described by A. F. Hess will answer, if a more elaborate refrigerator is not available.

Get from your grocer a deep box about 18 inches square, and put 3 inches of sawdust in the bottom. Place two pails in this box—one a smaller pail, inside the other —and fill the space between the outer pail and the box with sawdust. The nursing bottles filled with milk are placed in the inner pail. This pail is then filled with cracked ice, which surrounds the bottles. The inner pail should have a tin cover. Nail several thicknesses of

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[1] Helmholz, H. F.: The longitudinal sinus as the place of preference in infancy for intravenous aspirations and injections, including transfusion. Am. Jour. Dis. Child., x, 194, 1915.

[2] Unger, J. J.: A new method of syringe transfusion. Jour. Am. Med. Ass'n., lxiv, 582, 1915.

newspaper on the under surface of the cover of the box.
This ice-box should be kept covered, and in a shady, cool
place. The water from melted ice should be poured off,
and the ice renewed at least once each day.

Fig. 32.—Bed complete, with removable metal lid and
collapsible hood in place. The hood and lid can be re-
moved as indicated.

ELECTRIC-HEATED WATER-JACKETED INFANT BED.

This special bed, designed by the author is for use in
the care of premature and poorly nourished infants. The
advantages of this apparatus are:

1. Safety. The maximum temperature to which the
water can be warmed with the electric heater is about

155° F., with a room temperature of 70° F. and rheostat on contact 6, this giving a maximum temperature within the bed of about 110° F., with the lid and hood on.

2. Economy of operation, and, most important, the elimination of a trained attendant.
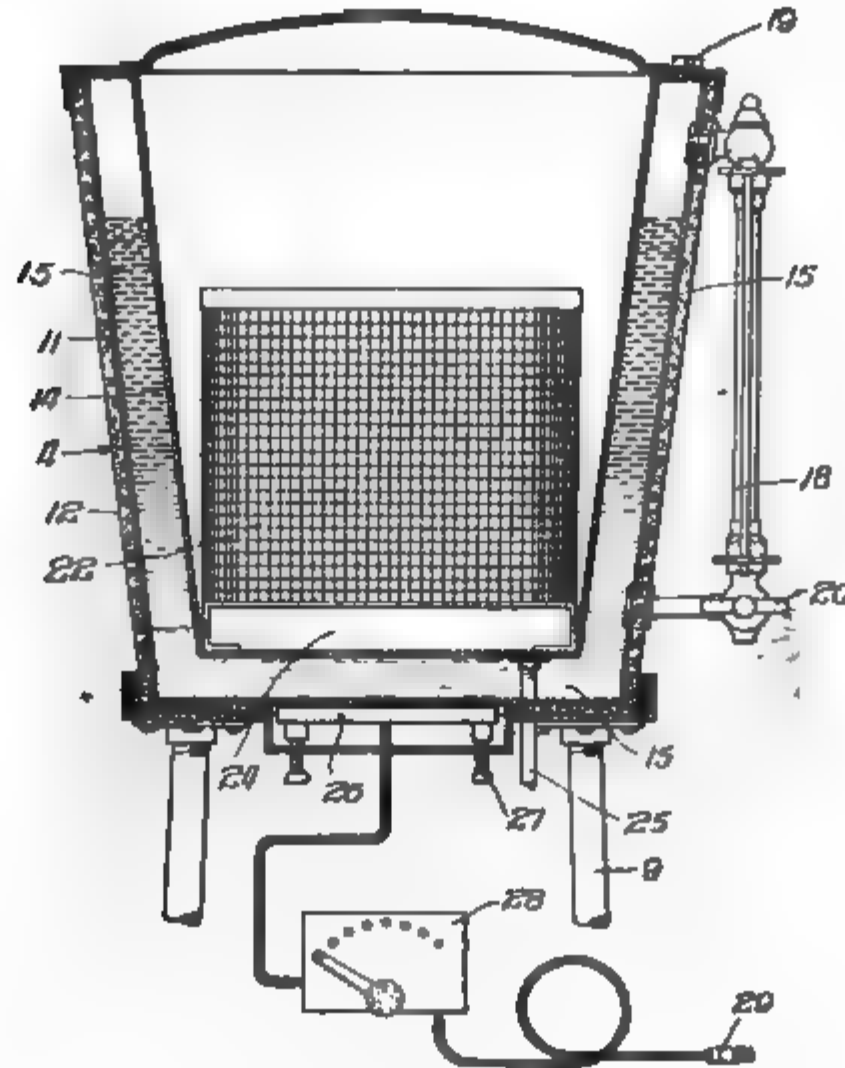
Fig. 33.—4, copper wall covering asbestos layer; 9, stand supporting bed; 11, 14, inner and outer walls of copper water jacket; 12, asbestos layer insulating water jacket. 15, water within jacket surrounding sides and floor of bed; 18, water gauge; 19, plug in opening used for filling jacket; 20, cock for emptying jacket; 22, removable crib; 24, air space underneath crib; 26, heating plate; 28, rheostat; 29, electric plug.

3. Simplicity of operation. It needs practically no attention unless there are extreme ranges of temperature

in the ward, since the asbestos insulation prevents radiation from the outer surface of the bed, and the heater holds the water at a constant temperature. It is seldom necessary to change the rheostat more than twice daily.

4. Ease of control of ventilation within the bed in the general wards of the hospital.　　　.

5. Humidity, which is little lower than that of surrounding air.

A COPPER JACKET TO PROTECT HEATING PADS.
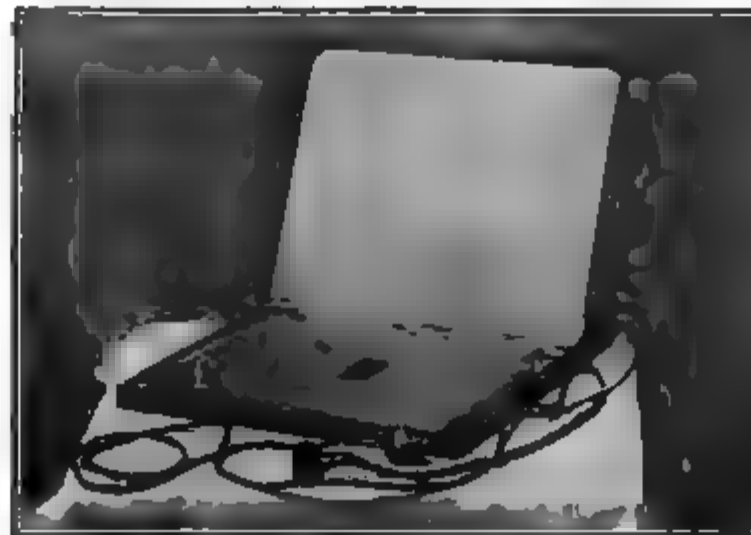
Fig. 34.—An asbesto ·lined copper receptacle for electric heating pads for use in the care of premature and debilitated infants (*Hess*). To avoid the danger of fire from short circuits in electric heating pads, a copper receptacle is used, 16 inches long, 13 inches wide, and 1¼ inches high, into which a 12 x 15-inch heating pad is laid. To allow of a maximum radiation from the lid or upper surface of the same, the floor and sides are lined with asbestos sheeting, while the lid is not lined. The cord passes through a small rubber insulator at the side to prevent contact with the metal and injury to the cord. This simple device can be used temporarily in wards and homes where better facilities for the care of this class of infants are lacking. It is to be placed in the bottom of a basket or crib, under the mattress or pillow.

CASE HISTORY.

(A) PRESENT ILLNESS.
1. Complaints: Mother's or patient's own statement.
2. Get history of present illness in detail: Onset, course and duration. Fever. Vomiting. Stools. Urine. Eruptions. Sleep, etc.
3. Previous treatment, if any.

(B) PREVIOUS HISTORY.
1. *Birth*: Para, nature and complications. ·
2. *Development*: Teeth (time of eruption), sat erect, walked, talked, mentality.
3. *General Health*: Robust or delicate, appetite, colds, fevers, coughs, bowels, convulsions, mouth-breathing, running ears, bed-wetting, etc.
4. *Illnesses*: Diseases similar to the present. Kind, date, duration, severity, recurrences, complications, careful history of acute infectious diseases.
5. *Feeding*: In detail in every infant.
   (a) *Breast* feeding: How long, intervals, condition of the baby, why discontinued.
   (b) *Artificial* feeding: Kind of food, intervals, how prepared, how much at each feeding, total quantity, how long used, effect on baby and on bowels, why discontinued.

(C) FAMILY HISTORY.
Parents, brothers and sisters.
(Constitutional diseases: Tuberculosis, syphilis, miscarriages (order of), rheumatism, nervousness or insanity, alcoholism.)

(D) EXAMINATION.
Examine patient fully.
1. *General appearance* and *weight*: Nutrition and general development, facial expression (intelligence, pain, etc.), amount of prostration, pallor, cry, nervous condition, posture, respiration.
2. *Skin*: Eruptions, turgor.

30

3. *Temperature*: Pulse and respiration (in infant omit temperature until 11).

4. *Head*: Size, shape, fontanelles (size, tension), cranio-tabes, eyes, nose (mouth, tongue, teeth under 12).

5. *Neck*: Goiter, glands, rigidity.

6. *Chest*: Shape, deformities, inequalities, expansions, *lungs* and *heart* in detail.

7. *Abdomen*: Size, distention, retraction, tenderness, rigidity, *liver, spleen, bladder, kidney,* fluid and tumors.

8. *Spine*: Deformities, rigidity.

9. *Genitalia* and *genital region*: Phimosis, vaginal discharge, fissures, inflammation, eruptions, hemorrhoids, pin-worms, etc.

10. *Extremities*: Glands, deformities, paralyses, atrophy, muscle tone, reflexes, athetosis, swelling, tenderness, discoloration, joints, gait.

11. *Temperature*: In child·under 3 years always rectal, and often in older children.

12. *Mouth*: Teeth, tongue, stomatitis, exanthemata, pharynx, tonsils, adenoids.

13. *Middle ear*.

14. *Special examinations*: Urine, blood, sputum, cultures, feces, vaccinations, serum reactions, etc.

AVERAGE WEIGHTS.

Age	Boys Pounds	Girls Pounds
Birth	7.55	7.16
Six months	16.50	15.50
Twelve months	20.50	19.80
Eighteen months	22.80	22.00
Two years	26.50	25.50
Three years	31.20	30.00
Four years	35.00	34.00
Five years	41.20	39.80
Six years	45.10	43.80
Seven years	49.50	48.00

AVERAGE WEIGHTS—*Continued.*

Age	Boys Pounds	Girls Pounds .
Eight years	54.50	52.90
Nine years	60.00	57.50
Ten years	66.60	64.10
Eleven years	72.40	70.30
Twelve years	79.80	81.40
Thirteen years	88.30	91.20
Fourteen years	99.30	100.30
Fifteen years	110.80	108.40
Sixteen years	123.70	113.00

MEASUREMENTS.

Age	Height in.	Chest in.	Head in.
Birth	20.5	13.25	13.75
6 months	25.0	16.0	17.0
1 year	29.0	18.0	18.0
2 years	32.5	19.0	18.75
5 years	41.5	21.0	20.5

Head at birth, 13.75 inches. First year, gain 4 inches; second year, gain 1 inch; 2 to 5 years, gain 1.5 inches for the 3 years.

Large head and small chest suggests rickets. The head is larger than the chest until second year, normally.

GENERAL DEVELOPMENT.

A healthy infant speaks single words toward the end of the first year, uses short sentences at the end of the second year; sits erect at the seventh month; stands with assistance at ninth or tenth month; attempts to walk at twelfth or thirteenth month, and walks freely at the fourteenth or fifteenth month.

SLEEP.

The healthy newborn infant sleeps practically all the time except when being fed.

	Hours per day
At birth	20 to 22
At end of 1st year	16 " 18
During 2d and 3d years	12 " 13
During 4th and 5th years	10 ' 11
During 12th and 13th years	8 " 9

ORDER AND AVERAGE TIME OF ERUPTION OF THE TWENTY DECIDUOUS TEETH.

	Months
2 lower central incisors	6 to 9
4 upper incisors	8 " 12
2 lower lateral incisors and 4 anterior molars	12 " 15
4 canines	18 " 24
4 posterior molars	24 " 30

At 1 year should have 6 teeth.
At 1 year 6 months should have 12 teeth.
At 2 years should have 16 teeth.
At 2 years and 6 months should have 20 teeth.

PERMANENT TEETH.

	Years
1st molars	6
Incisors	7 to 8
Bicuspids	9 " 10
Canines	12 " 14
Second molars	12 " 15
Third molars	17 " 25

CLOSURE OF FONTANELS.

Posterior fontanel usually closes by the end of the second month. Anterior fontanel at the end of the first year is about 1 inch in diameter, and usually closes at the eighteenth month. Normal variations, from fourteen to twenty-two months.

AVERAGE DAILY QUANTITY OF URINE IN HEALTH.

	Ounces
1st 24 hours	0 to 2
2d 24 hours	⅓ " 3
3 to 6 days	3 " 8
7 days to 2 months	5 " 13
2 to 6 months	7 " 16
6 months to 2 years	8 " 20
2 to 5 years	16 " 26
5 to 8 years	20 " 40
8 to 18 years	32 " 48

AVERAGE RATE OF PULSE AND RESPIRATION.

	Pulse	Respirations
Birth	140	35 to 40
1 month	120	25 " 40
6 to 12 months	105 to 115	25 " 30
2 to 6 years	90 " 105	25
7 to 10 years	80 " 90	22 " 25
11 to 14 years	75 " 80	20

BLOOD-PICTURE IN HEALTHY CHILDREN.

	Newborn	Infants	Older children
Hemoglobin	110 per cent.	70 to 95 per cent.	65 to 95 per cent.
Erythrocytes	5 to 8 millions	4.5 to 5.5 millions	4 to 4.5 millions.

AVERAGE WHITE CELL COUNTS.

1. Healthy children between 1 and 15 years of age average between 7000 and 15,000 leucocytes, approximately the same as adults.

2. Polymorphonuclear neutrophiles increase gradually from 30 per cent. in the first year to about 70 per cent. in the fifteenth year.

3. Lymphocytes decrease from 60 per cent. in the first year to about 30 per cent. in the fifteenth year. (This represents combined (large and small) lymphocytes.)

4. The reversal of the percentages of neutrophiles and lymphocytes occurs usually about the sixth year.

5. Eosinophiles average between 4 to 6 per cent., but vary greatly in different children at the same ages.

6. Transitional cells average approximately 2 to 3 per cent., not varying greatly at the different ages.

7. Mast-cells, about 0.3 to 0.6 per cent. Frequently absent.

8. Large mononuclear neutrophiles, 1 to 3.3 per cent. About the same at different ages.

Stool symbols	Urine symbols
N = normal.	A = albumin.
S = soft.	S = sugar.
W = watery.	Ac = acetone.
F = fat-soap.	D = diazo.
M = mucus.	I = indican.
Bl = blood.	C = casts.
C = curds.	P = pus.
G = green.	Bl = blood.
	Ep = epithelium.

GRAPHIC RECORD SHEET.

A brief description of the clinical sheet used in our wards may be of value, as it answers both the needs of a history sheet and of a daily chart as well. The points illustrated by it are: A graphic relationship between the temperature, weight, quality, and quantity of food taken, and the end-results on the stools and urine. Also separate spaces are provided for complications which may influence the preceding under the heading of symptoms, together with spaces for treatment other than dietetic, energy value of foods, vomiting, blood examinations, tuberculin reactions, etc. The small figures 1-10 are used to make an electrical reaction curve in cases showing a spasmophilic diathesis.

SARAH MORRIS HOSPITAL FOR CHILDREN

DEPARTMENT OF PEDIATRICS

UNIVERSITY OF ILLINOIS — ₪₪₪ — COLLEGE OF MEDICINE

No.

Name _____ Address _____

Date _____ Sex _____ Age _____ Race _____

Diagnosis _____

Name _____

Doctor _____

History of Patient.

Duration, symptoms, onset, earliest symptoms and later developments _____

Previous History.

BIRTH: Pre? Nature and Complications _____

DEVELOPMENT: Teeth _____

Sat Erect _____ Walked _____ Talked _____

Mentally _____

GENERAL HEALTH: _____

PREVIOUS ILLNESSES

_____

FEEDING HISTORY

Breast _____ No. of Months _____ Interval _____

_____ Why discontinued _____

ARTIFICIAL FEEDING. In Detail _____

_____

FAMILY HISTORY

_____

PHYSICAL EXAMINATION

_____

_____

_____

_____

_____

_____

_____

_____

_____

_____

_____

_____

TEMPERATURE   **P**   **R**   **Weight**   **Height**

LABORATORY EXAMINATIONS

_____

_____

_____

_____

_____

TREATMENT

_____

_____

_____

_____

_____

ASSIGNED TO

SUBSEQUENT TREATMENT AND REMARKS

Date	Weight	

PHYSICAL EXAMINATION

Temperature_____Pulse_____Respiration_____   (Underline each word describing condition)

INSPECTION: Bright   Dull   Nervous   Phlegmatic   Apathetic

GENERAL CONDITION: Fat   Thin   Good   Fair   Poor

SKIN: Normal   Prickly Heat   Eczema   Scabies   Seborrhea

     Tissue Turgor   Urticaria   Impetigo   Pediculosis   Nails

MUSCLES: Biceps and Thighs   Firm   Flabby

HEAD: Normal   Bosses prominent_____ _____Fontanelles_____

     Cranio-tabes_____

EYES: Pupils equal   Unequal   React to light   Distance   Nystagmus

     Motions: Normal   Abnormal   Blepharitis   Strabismus

NARES: Clear   Crushed   Discharge_____, character_____

SINUSES: Maxillary_____Frontal_____Ethmoid_____Sphenoid_____

MOUTH: Normal   Open   Herpes   Stomatitis (type)

TONGUE: Normal   Moist   Dry   Injected

THROAT: Normal   Injected   Membrane (type)_____

TONSILS: Normal   Large   Buried   Cryptic   Inflamed Operated Stumps_____Adenoids_____

GLANDS: Normal   Enlarged   Ant.-cervical   Post-cerv.   Epitroclear Inguinal   Others

TEETH: No._____Good_____No. Decayed_____Approximation good poor. Alveolar abscesses_____

EARS: Normal   Perforation   right   left.   Discharge   right   left   character_____

NECK: Thyroid_____Other findings_____

CHEST: Normal   barrel   flat   funnel   pigeon   Rosary   Harrison's Groove

LUNGS:_____

_____D'Espine_____

HEART: Area of dullness_____cm to left of mid-sternal line

                             _____cm to right of mid-sternal line

Apex 4th   5th   6th space in mid-clavicular line

"   _____cm outside, inside, mid-clavicular line

Action: Regular   Irregular   Sounds: clear   impure

Murmurs: NONE

            presystolic   apex   _____

     Soft

            systolic   pulmonic   trans. to   _____

     Loud

            diastolic   aortic   _____

Blood Pressure_____

ABDOMEN: Normal   large   distended   tympanitic   tender

     Hernia   umbilical   inguinal   right   left

LIVER: Palpable_____Enlarged_____Boundaries in Mid. Cl.L._____

SPLEEN: Palpable   Yes   No   Size_____

KIDNEYS:_____Right_____Left_____

GENITALS: Phimosis   Circumcised   Undescended   Testicle   Hydrocele   Vaginitis

     Anus   Normal   Abnormal

EXTREMITIES: Deformity   Acquired   Congenital   Rickets

FEET: Arches   good   flat   pronated   Posture   good   poor   Gait_____

SPINE: Normal   Rigid   Curvature   Round Shoulders

REFLEXES: Patellar   Brudzinski   Oppenheim   Trousseau

     Kernig   Babinski   Chvostek

WEIGHT: Present_____Normal for height_____

LENGTH: Present_____Normal for weight_____

     Ant. sup. spine to vertex_____to sole_____

CIRCUMFERENCE: Head   Chest   Abdomen_____

LABORATORY AND SPECIAL EXAMINATIONS

URINE:	Color	Sp. gr.	Reaction	Albumen
	Sugar	Acetone	Diacetic Acid	
	Pus	Blood	Casts	

SPECIAL NOSE, THROAT, EAR and SINUS:

EYE:

RADIOGRAM OF CHEST, DIGESTIVE TRACT, etc.:

BLOOD:	W. B. C.	R. B. C.	Hemoglobin	Diff. Ct.		Coag. Time

Vaccination

Wasserman Reaction:

Throat Culture:

Von Pirquet: negative   positive   cm. areola

Vaginal Smear

Electrical Reactions: COC   AOC   Relation AOC to ACC

Shick Test:

Skin Test for Proteins.

Stool Exam. for Parasites.

Toxin Antitoxin:

SUMMARY OF DEFECTS FOUND

Underweight for height		pounds	per cent		
Carious Teeth	No.		alveolar abcesses		
Mouth Breather			Cryptic Tonsils	Rt.	Lt.
Enlarged Cervical Glands					
Otitis:	dry	discharging	R.	L.	

OTHER PATHOLOGICAL FINDINGS

RECOMMENDATIONS:

Feeding

Medical

Surgical

Hygiene

Social Service

CONSULTATIONS ORDERED:

Consultations reports received and operations performed

SOCIAL SERVICE REPORT

Neighborhood   Home

Bath tub   sanitary conditions   Income

Mother's attitude   Remarks

CHILD WELFARE CONFERENCE

Name_____ Date_____ No_____

Address _____

Telephone_____ House Flat Front Rear Floor _____

Guardian's Name_____ Relationship_____

Sex_____ Age_____ Date of birth_____

Diagnosis _____

_____

_____

Referred by _____

Clinician _____Cross Index. Yes_____ No_____

WHY IS CHILD BROUGHT TO THE CONFERENCE?   (Mother's answer)_____

_____

_____

_____

(Does she think it is normal?)_____

HISTORY OF PRESENT ILLNESS (Onset, duration progress):

_____

_____

_____

_____

_____

_____

_____

FAMILY HISTORY

Name	Birthplace	Living	Dead	Age	Condition of Health	Cause of Death
Father						
Mother						

Children No.	Duration Delivery	Condition of Health	Age	Age at Death	Cause of Death

Miscarriages No.	Order of Pregnancies	Months	Cause

PERSONAL HISTORY

Full-term_____Premature at_____months   Birth Wgt_____

Condition at birth_____

Teeth: First at_____mos.   Sat erect at_____mos.   Walked at_____months.

Talked: Simple words at_____mos.   Short sentences_____mos.

Mental development_____

PAST ILLNESSES:

Diarrhea	Rickets	Bronchitis	Mumps	Pertussis	Pyelitis	Rheumatism
Snuffles	Spasmophilia	Pneumonia	Scarlet Fever	Chicken Pox	Scurvy	Chorea
Colds	Convulsions	Otitis	Influenza	Eczema	Acidosis	Endocarditis
Adenitis	Tonsillitis	Measles	Diphtheria	Enuresis	Syphilis	Nephritis

Operations _____

Important details and other diseases _____

_____

_____

PHYSICAL EXAMINATION

Temperature_____Pulse_____Respiration_____   (Underline each word describing condition.)

INSPECTION: Bright   Dull   Nervous   Phlegmatic   Apathetic

GENERAL CONDITION: Fat   Thin   Good   Fair   Poor

SKIN:

    Normal   Prickly Heat   Eczema   Scabies   Seborrhea
    Tissue Turgor   Urticaria   Impetigo   Pediculosis   Nails

MUSCLES: Biceps and Thigh   Firm   Flabby

HEAD: Normal   Bosses prominent_____Fontanelles _____

        Cranio-tabes _____

EYES: Pupils equal   Unequal   React to light   Distance   Nystagmus
        Motions: Normal   Abnormal   Blepharitis   Strabismus

NARES: Clear   Crusted   Discharge_____ character_____

SINUSES: Maxillary_____Frontal_____Ethmoid_____Sphenoid_____

MOUTH: Normal   Open   Herpes   Stomatitis (type)

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GLANDS: Normal   Enlarged   Ant.-cervical   Post-cerv.   Epitroclear Inguinal   Others

_____

TEETH: No_____ Good_____ No. Decayed_____ Approximation good poor. Alveolar abcesses_____

EARS: Normal Perforation right left. Discharge right left character_____

NECK: Thyroid_____Other findings_____

CHEST: Normal   barrel   flat   funnel   pigeon   Rosary   Harrison's Groove

LUNGS: _____

_____

_____ D'Espine_____

HEART: Area of dullness_____cm to left of mid-sternal line

                    _____cm to right of mid-sternal line

    Apex 4th   5th   6th space in mid-clavicular line

                    _____cm outside, inside, mid-clavicular line

    Action: Regular   Irregular   Sounds: clear   impure
        Murmurs: NONE
                    presystolic   apex   _____
                Soft
                    systolic   pulmonic   trans. to _____
                Loud
                    diastolic   aortic   _____

_____

_____Blood pressure _____

ABDOMEN: Normal   large   distended   tympanitic   tender
            Hernia   umbilical inguinal   right   left

_____

_____Blood Pressure_____

LIVER: Palpable_____Enlarged_____Boundaries in Mid. CLL._____

SPLEEN: Palpable   Yes   No   Size_____

Kidneys: Right_____   Left_____

GENITALS: Phimosis   Circumcised   Undescended Testicle Hydrocele Vaginitis
         Anus   Normal   Abnormal

EXTREMITIES: Deformity   Acquired   Congenital   Rickets

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SPINE: Normal   Rigid   Curvature   Round Shoulders

REFLEXES: Patellar   Brudzinski   Oppenheim   Trousseau
       Kernig   Babinski   Chvostek

WEIGHT: Present_____Normal for height_____

LENGTH: Present_____Normal for weight_____

        Ant. sup. spine to vertex_____to sole _____

CIRCUMFERENCE: Head   Chest   Abdomen_____

DIET AND HYGIENE.

Appetite (Especially for Breakfast'   good   fair   poor
Breakfast   Between   Dinner   Between   Supper   Bed-Time
Regularity of meals—hours

Food dislikes _____

Food Idiosyncrasies (milk, cereals, meat, eggs, vegetables, fruits) _____

No. of children in family_____Ages_____

Milk purchased daily	Rest periods yes no when_____
Milk taken daily	Sleep quality sound restless
Cereal	quantity hours
Vegetables potatoes other	No. sleeping in room in bed_____
Fruit	Fresh air window open closed
Meat or fish or eggs	Out or doors Hours
Coffee	Teeth How often brushed
Tea	Bath
Candy How much When eaten	Constipated yes or no Use of cathartics

LABORATORY AND SPECIAL EXAMINATIONS

URINE: Color   Sp. gr.   Reaction   Albumen
          Sugar   Acetone   Diacetic Acid
          Pus   Blood   Casts

SPECIAL NOSE, THROAT, EAR and SINUS: •

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RADIOGRAM OF CHEST, DIGESTIVE TRACT, etc.:

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Mouth Breather   Cryptic Tonsils   Rt.   Lt.
Enlarged Cervical Glands
Otitis: dry   discharging   R.   L.

OTHER PATHOLOGICAL FINDINGS_____

_____

_____

RECOMMENDATIONS:
Feeding

_____

_____

_____

_____

_____

Medical

_____

_____

_____

Surgical

_____

Hygiene
Social Service

CONSULTATIONS ORDERED:
Consultations reports received and operations performed_____

_____

_____

SOCIAL SERVICE REPORT

Neighborhood   Home

Bath tub   sanitary conditions   Income

Mother's attitude   Remarks_____

_____

_____

SUBSEQUENT TREATMENT

DATE	TEMP.	WGT	

SUBSEQUENT TREATMENT

Date	Age	Weight	Temp.	

INDEX.

(485)